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VOL. XXXIII OCTOBER, 1923 No. 10

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

FOUNDED IN 1896 BY
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Subscription, $6.00 per Annum, in Advance.

Foreign Subscription, 35 Shillings per Annum, Post Free.
Single Copies, 75 cents.

PUBLISHED BY THE LARYNGOSCOPE CO.
3858 Westminster Place, St. Louis, Mo., U. S. A.

FOREIGN OFFICE, BAILLIERE, TINDALL & COX,
8 HENRIETTA ST., STRAND, LONDON, ENG.

(Entered at the Postoffice at St. Louis, Mo., as Second-Class Matter, in July, 1896.)

The first four numbers of this new quarterly have been issued. Subscribe now.

Vol. 2 JULY, 1923 No. 3.

ORALISM AND AURALISM

A QUARTERLY JOURNAL DEVOTED EXCLUSIVELY TO PROBLEMS OF THE

DEAF AND DEFECTIVE SPEECH

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Single Copies, 50 cents.

PUBLISHED BY

THE LARYNGOSCOPE CO., ST. LOUIS.

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LARYNGOSCOPE.

Vol. XXXIII ST. LOUIS, OCTOBER, 1923 No. 10

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE PATHOLOGY OF DEAFMUTISM.*

DR. J. S. FRASER, Glasgow, Scotland.

I have been asked to present the subject of the Pathology of Deafmutism. It is not possible for me to go fully into the whole subject, but I can attempt to lay before you an account of the principal pathological conditions which result in such a severe degree of deafness that the child becomes a deafmute. Cases of deafmutism are usually divided into (I) Congenital, and (II) Acquired. This is not scientifically accurate. Congenital deafness usually includes not only "developmental" cases due to faults of the germ plasm, but also those due to such inflammatory conditions as intrauterine meningitis. In other words, "congenital" has been regarded as synonymous with "before birth." Cases due to congenital syphilis are difficult to classify; they do not, as a rule, become deaf until childhood is well advanced. It appears to be more scientific to divide cases of deafmutism into (I) those due to an error in development and (II) those due to inflammatory conditions. The main causes of deafmutism may therefore be classified as follows:

(*I*) *Congenital or Developmental Deafness*: (A) Endemic. (B) Sporadic—(1) Aplasia of the Whole Labyrinth; (2) Cases in which both the Bony and Membranous Labyrinths are affected; (3) Congenital Malformation affecting both Cochlear and Vestibular Apparatus; (4) Sacculo-Cochlear Degeneration; (5) Cases in which the Membranous Cochlea alone is affected.

*Address presented by invitation before the American Otological Society, May 15, 1923, at Atlantic City, N. J.

(*II*) *Acquired or Inflammatory Deafmutism,* due to (A) Traumatism; (B) Labyrinthitis following Otitis Media; (C) Meningitic Neuro-labyrinthitis; (D) Congenital Syphilis.

I. Congenital or Developmental Deaf-Mutism (Constitutional Deafmutism or Hammerschlag).

(*A*) *Endemic or Cretinic Deafness (Siebenmann's Type.)*

Clinical Aspect. Patients may be mentally feeble; a few are bright. Most of the cases are complete cretins. Goitre may be present. The majority of the patients are deaf from birth. Some of Nager's cases showed sound-conduction deafness. Stein states that 25 per cent of cretins have normal hearing, 45 per cent slight deafness, 25 per cent severe, and 5 per cent absolute deafness. Nager mentions the broad-based, waddling gait of his patients and attributes this to changes in the brain.

Anatomical Changes:—

Middle Ear. Alexander mentions myxomatous thickening of the submucous tissues of the middle ear, with filling up of the window niches by connective tissue and fat cells. In many, the long process of the incus and the head of the stapes are adherent to the facial canal. Bony ankylosis of the footplate of the stapes to the margin of the oval window is mentioned by several writers. Exostoses are frequently found on the promontory. The tympanum may be obliterated by new-formed spongy bone.

The Labyrinth Capsule. Alexander and Nager have noted areas of otosclerosis in the labyrinth capsule in cases of cretinic deafness. Endostoses have also been observed growing from the labyrinth capsule into the hollow spaces of the cochlea, vestibule and canals.

Inner Ear. Habermann, Schlitter, and many others have found the inner ear *normal.* Few writers have reported any gross abnormality. Alexander however, found marked degenerative changes in Corti's organ.

Manasse thinks that hardness of hearing is produced by changes in the middle ear, especially in the window niches, but complete deafness comes on when labyrinth atrophy occurs. The middle ear changes are due to the failure of the normal resorption of the foetal connective tissue. Stein groups endemic deafmutism along with sporadic congenital deafmutism (Hammerschlag's hereditary degenerative deafness), and includes them both under the heading of "constitutional" deafness.

Siebenmann and Nager take the view that in many cases the deafness is the result of changes in the brain. Denker holds that the

condition results from disorders of metabolism owing to loss of thyroid secretion.

(*B*) *Sporadic Congenital Deafness.*

(*1*) *Aplasia of the Whole Labyrinth.* (*Michel's Type*). In Michel's case there was a total defect of both labyrinths and eighth nerves, while the external meatus and tympanic membranes were normal. Siebenmann explains this condition by postulating absence of the otic vesicle in the first month of foetal life. Schwartze has reported a similar case. Virchow and Delille have recorded cases of complete absence of the petrous pyramid. Ogston has published in the case of a child who suffered from congenital atresia of the external meatus on one side, with atrophy of the eighth nerve, hemiatrophy of the cerebrum and cerebellum and hypoplasia of the petrous pyramids.

(*2*) *Both the Bony Membranous Labyrinths may be affected.* (*Mondini's or Alexander's Type.*) Alexander's case was completely deaf. Alt's patient was deaf from birth, and a sister was also deaf.

Pathology Bony Labyrinth. In Mondini's case, the whole bony capsule of the cochlea was flattened from base to apex as in certain of the lower mammals. A more or less normal arrangement of the scalae was only present at the lower part of the cochlea. In the upper part there was a wide common space. Alexander points out that a similar condition is seen at the second month of foetal life and holds that in these cases development stops at this period. Alexander mentions atrophy of the maculae of the utricle and saccule.

In Mondini's and Schoenemann's cases the aqueduct of the vestibule was dilated and formed a gutter (A similar condition has been noted by Gray in certain of the lower mammals.)

The cochlear canal may be collapsed or dilated. Corti's organ may be absent, or embryonic. Alexander mentions that in his case the cochlear ganglion did not take a spiral course but was situated centrally in the modiolus. He believes that the faulty formation of the septa between the various scalae is associated with the anomalous position of the spiral ganglion. The condition of the cochlear nerve coincides with that in the lowest mammals—duckmole and echidna.

Siebenmann attributes the condition to some abnormality occurring at the fourth month of embryonic life. The development of the cochlea takes place from base to apex, so that, should the development be arrested at a certain point, the septa between the various

coils may fail to form, with the result that the scala vestibuli of one coil communicate with the scala tympani of the next higher coil.

(*3*) *Congenital Malformation affecting both the (Membranous Cochlear and Vestibular Apparatus)*. This clinical type of deafmutism is frequently associated with retinitis pigmentosa and mental deficiency. Not only is there complete or almost complete deafness but the vestibular reactions are absent or at least greatly diminished. Bezold's two cases showed uncertain gait. Of the four ears (two cases) one was quite deaf, whereas the other three showed a hearing island toward the middle of the scale. (In retinitis pigmentosa there is a contraction of the visual field corresponding to the contraction of the hearing field in these deafmute cases. Frey and Hammerschlag found that in four cases the semi-circular canal apparatus failed to react to rotation; two of the patients had deafmute imbecile brothers or sisters.

Siebenmann and Bing's case was the fourth of eight children, of whom four were apparently born deaf. A brother also had retinitis pigmentosa. The galvanic reaction was absent.

Pathology:—

Vestibular Apparatus:— Siebenmann and Bing found the maculae and cristae degenerated but the vestibular nerve and ganglion normal. There was marked atrophy of the vestibular nerve and ganglion in both of Oppikofer's cases.

Cochlear Apparatus. Nager found the membranous cochlea dilated or collapsed and the sensory epithelium absent or badly developed. The spiral ganglion was atrophied in both of Oppikofer's cases. Nager and also Hane found the stem of the cochlear nerve markedly atrophied. The vascularity of the nerve endings in the cochlea and vestibule were poor. (This corresponds to the pathological changes in retinitis pigmentosa.)

(*4*) *Sacculo-Cochlear Degeneration. (Scheibe's Type)*. (The case described in this paper belongs to the Scheibe Type.) About 70 per cent of cases of congenital deafmutism belong to the "sacculo-cochlear" type, in which the utricle and canals are intact. Hearing remnants are usually present. The vestibular apparatus reacts normally or almost normally to the rotation and caloric tests. In Oppikofer's case the parents of the patient heard well. They had seven sons and one daughter, and of these, three, including the patient were deaf from birth. Oppikofer's patient who was stone-deaf, married a deafmute woman whose brother was also a deafmute. The marriage produced one daughter, who was deaf, but had a normal vestibular apparatus.

Pathology:—As a rule no changes are found in the middle ear. If present, these are merely accidental (as in the case illustrated).

The bony labyrinth is usually normal, but in Lindt's case there was on one side an otosclerotic focus. Lindt holds that this had nothing to do with the patient's deafmutism. Gray has investigated four cases and found that the hollow spaces of the bony labyrinth were larger than normal.

Utricle and Canals. The neuro-epithelium of the utricle and cristae of the canals are normal in almost all cases.

Sacculus. In Scheibe's original case there were no hair cells evident in the sacculus. In Lindt's, Goerke's and Quix's cases the sacculus was almost completely collapsed and the neuro-epithelium absent. The ductus reuniens were obliterated in many cases.

The Cochlear Canal may be (1) collapsed in its entirety, or (2) dilated; (3) or it may be dilated at one part and collapsed at another. The dilatation may be so great that the scala vestibuli is obliterated. If the canal is collapsed Reissner's membrane is usually adherent to the remains of Corti's organ or to the basilar membrane.

Corti's organ may be absent in parts or unrecognizable. It may be replaced by cubical or rounded cells. In Scheibe's second case Corti's organ was almost normal in the upper coils. Hyaline bodies are often present in the cochlear canal, and in some there is a large amount of pigment present.

The membrana tectoria is usually covered with epithelium and tucked into the internal spiral sulcus, or backwards on to Huchke's tooth. In other cases the membrana tectoria is attached across the scala media to the stria vascularis and covered on both surfaces with a layer of epithelium.

The stria vascularis may be absent or replaced by hyaline masses. The stria formed a polypoid projection into the scala media in at least two of Gray's four cases.

The spiral ganglion and the fine branches of the cochlear nerve usually show atrophy. The stem of the cochlear nerve was atrophied on both sides in Scheibe's first case.

(5) Here the changes are confined to the Membranous Cochlea and Nerve. The sacculus and the pars superior are healthy. The changes in the scala media are the same as those already described under Scheibe's type (4).

A Case of Congenital or Developmental Deafmutism.

(See also Journal of Laryngology and Otology, 1922, p. 13)

K. McD., male, aged 11 years. The parents stated that the patient had been born totally deaf but his teacher in the oral class regarded

him as having slight remains of hearing. For several years the boy had suffered from foul-smelling otorrhea (left). On admission, pain and mastoid tenderness were present; temperature 102.5°; conversation voice not heard by either ear; C.128 apparently heard by right ear but not by left; C.256 was heard by both ears, and the tone correctly imitated by the boy. Cold syringing of each ear produced distinct rotary and lateral nystagmus to the opposite side. Death was due to purulent meningitis. Post-mortem twelve hours after death.

Microscopal Examination of Left (Operated) Ear.

Tympanic Cavity. There is a great swelling of the sub-mucosa, with small cell infiltration. Cholesteatoma and pus are present in the antrum. The prominence of the lateral canal is eroded. The mucosa of the oval window niche is swollen. The round window membrane is normal. *Vestibule*: The neuroepithelium of the utricle is healthy, but the otolith membrane is separated (artefact?). The sacculus is collapsed (Fig. 5) but the epithelium is well formed in parts. The duct from the saccule to the endolymphatic duct is narrow. *Cochlea*: Reissner's membrane passes vertically upward, so that the cochlear canal is dilated. (Fig. 1-4) Corti's organ is badly formed; nowhere is the normal acoustic papilla visible. It is best developed in the apical portion. The pillar cells, though deformed, can be made out in parts. The membrana tectoria is enclosed in epithelium; in the basal coil it is tucked into the inner spiral sulcus. (Fig. 4). In the upper part of the basal coil it is attached to the stria vascularis; (Fig. 3) in the middle coil it is turned inwards towards the modiolus and helicotrema; (Fig. 2) the spiral ligament shows clear areas (Fig. 4)—so-called "dropsical degeneration." The stria vascularis at the apex of the cochlea is almost absent. The cochlea nerve itself is well formed (Fig. 1) and the ganglion cells are present. *Canals*: The cristae of all the canals show slight desquamation of the epithelium and separation of the cupula—possibly associated with the high temperature before death. (Fig. 6). *Internal Meatus*: Meningitis is present. The vestibular ganglion cells are present in normal number.

Microscopical Examination of right (non-operated) Ear. The *middle ear* is healthy, except for thickening of the mucosa and small cell exudation in the niche of the round window.

Vestibule: The sacculus is collapsed and the ductus reuniens obliterated. The utriculus is normal. (Fig. 12).

Cochlea: A few leucocytes are seen in the scala tympani in the upper surface of the round window membrane. They have evident-

ly come from the subarachnoid space along the cochlear aqueduct. The cochlear duct itself is narrow or collapsed. (Figs. 7-11). Reissner's membrane is bent downwards, and is adherent to the overgrown stria vascularis for at least half its extent. (Figs. 8, 9 and 11). In the apical coil the scala media is quite filled up, (Fig. 8) and is adherent to the bony partition between the apical and middle coils. Corti's organ is for the most part an irregular heap of cells. (Fig. 10). The membrana tectoria is adherent to Corti's organ, and also to the stria vascularis. In places the membrana tectoria is tucked into the internal spiral sulcus. (Figs. 9 and 10).

The Canals are healthy.

Internal Meatus: There are many pus cells and some blood in the fundus of the internal meatus between the arachnoid and dura mater (Fig. 7) (basal meningitis). The cochlear nerve shows quite the normal thickness. The vestibular ganglion cells are not diminished.

Remarks: The case is one of "congenital or developmental" deafness belonging to the Scheibe type, with slight remains of hearing and a normal or almost normal vestibular reaction. It is of interest that on the right side the cochlear canal is collapsed, especially at the apex, while on the left side it is dilated. This condition has been noted by previous observers.

Explanation of the Changes found in the Scheibe type of Congenital or Developmental Deafmutism.

Various theories have been advanced to account for the abnormalities observed. (1). Faulty Development. Oppikofer suggests that the otic vesicle may have been adnormally large, while the cartilaginous cochlea, which develops later, was of normal size. This may account for the fold formation. As an alternative, he suggests that the fold formation may have been due to adhesion of the walls of the cochlear canal. If the development disturbance occurs in the first two months of foetal life the membrana tectoria is not formed. In such cases the fine nerve branches and the spiral ganglion remains markedly undeveloped. If the developmental disturbance occurs later the membrana tectoria may be properly formed and the ganglion and nerves well developed.

(2) Hereditary Weakness of the Ear. Manasse and also Siebenmann have called attention to the similarity of the changes found in congenital deafness to those seen in chronic progressive labyrinthine deafness. Manasse believes that the atrophic degenerative process may occur in intra-uterine life. Politzer was the first to describe progressive labyrinthine deafness coming on in youth and re-

sulting in marked or even complete deafness. He thought that the condition was due to an idiopathic atrophy of the acoustic nerve. Spira has called attention to the occurrence of ear disease—otitis media or nerve deafness—in certain families. He regards these contions as due to an inherited local lack of resistance. Gowers in 1902, and Edinger in 1904, called attention to the condition of "abiotrophy," in which there is a want of nervous energy to bear the strain of normal or excessive functional activity. Such conditions as progressive muscular atrophy, primary optic atrophy, and progressive nerve deafness belong to this category.

(3) Increased Pressure in Perilymphatic or Endolymphatic Spaces. Gray thinks that increased pressure in the perilymphatic space transmitted from the cranial cavity through the aqueduct of the cochlea might account for the depressed condition of Reissner's membrane. Dilatation of the cochlear canal, on the other hand, may be due to increased secretion of endolymph from the enlarged stria vascularis. Quix believes that the explanation of the deafmutism is to be found in the stria vascularis, which is a secretory organ, and regulates the supply of endolymph. Pritchard considers that the hypertrophied condition of the stria resembles very closely the tegmentum vasculosum in the cochlea of birds.

(4) Inflammatory Changes: Meningitis. Siebenmann, Nager, and Oppikofer believe that the conditions present were due to an intra-uterine inflammatory process. Meningitis is very frequent during the first year of life. Preysing has recorded two cases of otitis interna in sucklings. Seibemenn mentions the possibility of placental infection. Gradwohl of St. Louis has recorded the case of a woman, aged 31, seven months pregnant, who suffered from pain in the left ear rapidly followed by coma. Well-marked signs of meningitis were present. The patient died undelivered. Autopsy revealed acute nephritis and purulent basal meningitis. The fetus also showed sero-purulent meningitis. Bacteriological examination of the cerebro-spinal fluid of both mother and fetus revealed the *diplococcus intra-cellularis,* and the same organism was separated in pure culture from the left ear of the mother. So far, however, there is only the case of Haike which goes to prove the existence of an intra-uterine meningitic labyrinthitis.

The writer does not believe that sporadic congenital deafmutism is due to intra-uterine meningitis; firstly, because the membranous labyrinth is developed from the epiblast and only later becomes connected to the central nervous system by means of the acoustic ganglia and eighth nerve; secondly, because it is difficult or impossible to

think of foetal meningitis affecting only the saccule and cochlea and leaving intact the remaining parts of the membranous labyrinth; and, thirdly, because the changes observed in congenital deafness differ so markedly from those produced in the inner ear in cases of meningitic labyrinthitis in post-fetal life.

II. Acquired or Inflammatory Deafmutism.

The pathology of acquired deafmutism is the pathology of labyrinthitis occurring in intra-uterine or post-fetal life. Intra-uterine labyrinthitis is almost certainly of meningitic origin. Post-fetal labyrinthitis, on the other hand, may be due (A) to traumatism, as in fracture of the cranial base; (B) to otitis media with invasion of the labyrinth through the oval or round windows or through the prominence of the lateral semi-circular canal; or (C) to purulent leptomeningitis with infection of the inner ear either along the fibres of the eighth nerve or along the aqueduct of the cochlea. It must be admitted that it may be difficult to determine by microscopic examination—many years after the occurrence of deafness—the exact route of the infection of the labyrinth. In other words, we cannot in many cases be certain whether the labyrinth was invaded from the middle ear or from the meninges. (D) Congenital syphilitic changes in the ear.

(*A*) *Deafmutism due to Trauma.* Deafmutism due to this cause follows fracture of the cranial base, which involves the labyrinth on both sides. Unless the injury to the labyrinth occurs during the first ten years of life, deafmutism is not produced.

Katz reports the case of a man, aged 50, who at the age of three years suffered from a head injury, followed by loss of consciousness for several days. On recovery it was found that the patient was deaf. Microscopic examination showed that the hollow spaces of the labyrinth were filled with spongy bone. Bony ankylosis of the stapes was present on the left side. There was marked atrophy of the nerve structures of the whole labyrinth.

Nager's patient was a man, aged 64. As a child of 4 years he received a severe head injury and lost his hearing. On microscopic examination both eighth nerves were atrophied. The labyrinth capsule showed traces of old fracture involving the region of the sacculus and round window. The fracture had apparently been a vertical one at right angle to the long axis of the petrous pyramid. The cochlea, vestibule and canals showed new bone formation. The cochlear duct, utricle and saccule were dilated. Corti's organ showed atrophic degeneration of the neuro-epithelium. Only a few shrunken cells of the spiral ganglion remained.

(*B*) *Deafmutism due to Labyrinthitis following Middle Ear Suppuration.* Denker states that middle-ear lesions alone do not as a rule produce such severe deafness as to give rise to deafmutism. He believes, however, that closure of both labyrinth windows may produce deafmutism even when the inner ear is intact. In severe cases of *scarlatinal* otitis media the tympanic ossicles and joints are destroyed and the superficial layers of the tympanic walls are necrotic Invasion of the labyrinth may occur through the oval or round windows. Of three cases of deafmutism resulting from scarlatina, in which the labyrinth was examined, chronic suppurative labyrinthitis was found in the first, new connective tissue and bone formation in the second, while the third the hollow spaces of the labyrinth were filled with cholesteatoma, which had passed in through the open oval window. In *deafmutism due to measles* almost the same marked changes have been found in the middle ear and labyrinth.

CASE OF ACQUIRED OR INFLAMMATORY DEAFMUTISM.

(See Journal of Laryngology and Otology, 1922, p. 57).

C. S., boy, aged 10 years, was admitted to the Royal Infirmary on January 5th, 1917 from a Deaf and Dumb Institution. Few details were obtainable as the mother was in prison, and was only seen after the death of the patient, and the father was absent on active service. The mother herself was rather deaf and suffered from tinnitus. Of her twelve children only the patient was deaf. He had never spoken and was late in learning to walk, two years and eight months. Discharge from the ears only began two years before admission to hospital.

On examination of the right ear an attic perforation was observed, from which some cholesteatoma protruded. The left drumhead was retracted and showed an adherent scar in the posterior superior part. Functional examination was impossible. The radical mastoid operation was advised but permission could not be obtained from the parents. On June 20th, the patient was admitted as an urgent case for operation. About June 16th, the boy had began to be feverish, the temperature rising to 102° F. each afternoon. *Examination.* A polypus was present in the right external meatus. The patient did not respond to vowels spoken in a loud voice close to his right ear. No Rombergism, no spontaneous nystagmus, no fistula symptom; rotation produced no nystagmus. The cold caloric test was negative on both sides. The temperature rose to 106° F. at 8 p. m. but there was no shivering or vomiting. On June 21st, the Medical Managers of the Infirmary decided that operation should be performed at once in spite of the lack of permission

from the parents. *Operation.* The cerebro-spinal fluid was clear but under tension. An extradural abscess was evacuated. The sinus was found to be thrombosed for two inches backwards from the upper knee. The internal jugular vein was ligated and divided. Later, intravenous injections of eusol were tried, but the rigors continued and metastatic abscesses developed in the left lung, with empyema. The patient died three weeks after operation.

Microscopic Examination of the Right (operated) Ear.

Middle Ear Cleft. Bony Eustacian tube practically obliterated by radical mastoid operation (Fig. 13); mucosa of tympanum and antrum thickened and infiltrated with small cells; cholesteatoma on the inner wall of the attic; marked irregularity of the bone over the prominence of the lateral canal (Fig. 14 and 15); no evidence of a former perforation of the oval windows; mucosa of the round window niche thickened (Fig. 17).

Cochlea. There is new connective tissue and bone formation in the scala vestibuli (Fig. 13 and 16), which is practically obliterated. New bone formation is present in the scala tympani of the basal coil (Fig. 17), but in the upper coils this scala is practically normal. The cochlear canal is greatly dilated in all coils. (Fig. 16). Corti's organ is merely a low layer of cells. The membrana tectoria does not reach Corti's organ. The cells of the spiral ganglion are reduced and the bony spiral lamina contains a few nerve fibres. The perilymphatic aqueduct is obliterated at the cochlear end (Fig. 17).

Vestibule. The saccule and utricle are greatly dilated and the neuro-epithelium degenerated. New bone formation is present at the point where the scala vestibuli opens into the vestibule (Fig. 13).

Canals. At its convexity and towards the smooth end the lateral canal is quite obliterated by new bone formation (Fig. 14 and 15). The crista is atrophied. The condition of the superior and posterior canals is similar.

Internal Meatus. The nerves and vestibular ganglion appear normal.

Microscopic Examination of the Left (non-operated) Ear.

Middle Ear Cleft. The posterior part of the drumhead shows a retracted scar (Fig. 18), which is adherent to the long process of the incus. On the left side there is considerable thickening of the bone over the prominence of the lateral canal. The mucous membrane of the round window is greatly thickened (Fig. 18). The tympanic ossicles, joints, and muscles are normal.

Labyrinth. The changes in the labyrinth are similar to but rather more marked than, those found on the right side (Fig. 19, 20, 21, 22).

Remarks: The case is undoubtedly one of acquired or inflammatory deafmutism resulting from suppurative labyrinthitis. It is not possible to be dogmatic as to the route of infection. The labyrinth may have been invaded on both sides from the middle ear, through the prominence of the lateral canal. The fact that the changes were most marked in the canals, and that in the cochlea they were more pronounced in the scala vestibuli than in the scala tympani, point in this direction. On the other hand, it must be admitted that meningitis labyrinthitis is a possible explanation of the condition. Intrauterine meningitis cannot be excluded in view of the history that the child was late in learning to walk and had never been able to speak. Reliance cannot be placed on the statements of the boy's mother, who asserted that middle ear suppuration had only commenced just before admission to the Deaf and Dumb Institution, because cholesteatoma was found at operation upon the right ear and a retracted scar on examination of the left drumhead.

II. (C) Deafmutism due to Neuro-Labyrinthitis following Purulent Meningitis.

(Transactions of Otological Section Royal Society of Medicine, January 16th, 1920).

The most frequent cause of this condition is epidemic cerebrospinal meningitis. Measles and pneumonia may be followed by meningitis and labyrinth suppuration. "Mumps" deafness and osteomyelitic deafness may have the same pathology. The nerve deafness may have the same pathology. The nerve deafness of secondary syphilis is closely associated with specific leptomeningitis.

The original site of infection may be the upper or lower respiratory tract, the parotid gland, the tonsil, the alimentary tract, or other part. In all cases, however, invasion of the blood-stream is probably the next step in the production of meningitis. We know that during an epidemic of "spotted fever" many people have meningococci in the nasopharynx for longer or shorter periods, and yet do not develop a blood infection and its consequent meningitis. Further, of those who develop meningitis only a certain percentage become deaf. We do not know, however, why the labyrinth is affected in these latter cases. We may, if we like, put it down to some inherent weakness of the auditory and vestibular apparatus.

Epidemic Cerebro-Spinal Meningitis.

Deafness is the gravest complication of this disease. It is worst in the severest cases, though it may occur in the benign or abortive varieties. As a rule the deafness comes on in the first or second week. Both ears are as a rule affected. Apparently the onset is sudden, but the patient is almost unconscious and does not complain. (Kerr notes that deafness is by no means the only complication; arthritis and orchitis may occur as a result of blood infection.) Of the patients who recover, from 20 to 37 per cent are deaf. Children as a rule become totally deaf and on recovery show a staggering gait (like the waddling gait of a duck) for some months or even a year after recovery. The older the patient the sooner these symptoms pass off.

Epidemic Cerebro-Spinal Meningitis as a Cause of Deafmutism.

Statistics on this point vary from 2 to 26 per cent. The discrepancy may be explained as follows: If there has been a recent out break of the disease in a country we have a high percentage of deafmutism due to epidemic meningitis, but if the country has been free from outbreaks for many years the proportion is low. Of thirty-four cases of deafmutism following "spotted fever" seen at Dr. Logan Turner's department during the years 1907-18 (inclusive), thirty were brought by the parents during the four years of 1907-10. Of 111 cases of deafmutism examined by the writer at the Edinburgh Royal Institution for the Education of the Deaf and Dumb, fourteen were due to meningitis. Of these, seven were cases of epidemic cerebro-spinal menigitis. Examination showed normal or only retracted drumheads in five out of the seven cases, while functional tests revealed absolute deafness and complete loss of vestibular reaction in six cases.

Pathology. The deafness may be due to (1) Hydro-cephalus; (2) purulent infiltration of the ependyma of the fourth ventricle; (3) Infiltration of the eighth nerve with purulent exudate; (4) Purulent labyrinthitis; (5) Otitis media.

The stages of neurolabyrinthitis due to epidemic cerebro-spinal meningitis appear to be as follows: (1) Purulent infiltration within the arachnoid nerve sheath in the internal auditory meatus. Deafness may occur either from changes in the nerve or from secondary descending degeneration. (2) Purulent invasion of the perilymphatic and, later, of the endilymphatic spaces of the cochlea, vestibule and canals, the invasion occurring along the nervous and vascular paths. The pus may burst outwards through the windows into the tympanic cavity, and so give rise to meningitic otitis media. (3)

After the above stage has passed, if the patient lives long enough, there is formation of granulation tissue within the labyrinth. (4) Later on we have the formation within the labyrinth of new connective tissue and bone which partly or completely fills the hollow spaces of the inner ear. (5) The middle ear may be involved by way of the Eustachian tube. Opinions as to the frequency of otitis media vary greatly; thus Netter states that it is not common and is only found in advanced cases. Altmann has examined twelve cases of deafness and found the middle ear unaffected. Goepport, on the other hand, found otitis media in 50 per cent of cases and in 62 per cent of post mortems.

Siebenmann has described the pathological changes in the acute stage of meningitic labyrinthitis. The process begins in the perilymphatic space with hyperaemia, stasis, thrombosis, and rupture of the smaller vessels. This is followed by infiltration and necrosis of the endosteum. Even the thin plate of endosteal bone may be loosened. The blood supply from the endosteum is cut off, and this leads to necrosis of the sensory epithelium. The eighth nerve at this stage is embedded in pus, though the labyrinthine fluid is still a blood-stained serum. Later on, it becomes purulent, with complete destruction of the membranous labyrinth. The next stage consists in the formation of granulation and connective tissue from the remains of the endosteal layer. Lastly there is new bone formation in the connective tissue.

Case I. Corporal G., admitted June 21, 1916. Patient had had violent headache for fourteen days, with cerebral vomiting. Photobia and opisthotonos present; temperature subnormal; pulse 40 to 60. Death on June 25, 1916.

The tympanic membrane is thickened and shows new bone formation ("chalk patch") (Fig. 23). There is new connective tissue between the ossicles and the outer wall of the attic. An adhesion stretches across from the anterior margin of the oval window to the tympanic membrane (Fig. 23). The long process of the incus and the crua of the stapes are absent. (There had evidently been a former chronic suppurative otitis media.) Corti's organ is not well formed; the pillar cells can only be recognized in the middle coil. *Internal meatus.* There is a little pus within the arachnoid sheath (Fig. 23), but there is no infiltration of the cochlear or vestibular nerves by menigitis.

Case II. Corporal D., admitted August 7, 1916. Patient lies on the right side in a semi-comotose condition. Kernig and Babinski signs present. *Patient has marked horizontal and rotatory nystagmus to the left* but is too ill for functional examination of the ear.

Post-mortem. Pus in subarachnoid space all over cerebrum. Adhesions and pus in interpreduncular space and cisterna points. There is a mass of purulent exudate round the nerves on the right side, from the 5th to the 12th. Foramen of Magendie closed by recent adhesions. Ventricles contain thick pus. The brain shows numerous petechial hemorrhage. The nasal accessory sinuses are normal. The left (middle and inner) ear is normal.

Microscopic Examination of the Right Middle and Inner Ear. The *middle ear* spaces are healthy, except in the neighborhood of the oval and round windows where there are a few pus cells and some fibrin (Fig. 25). The anterior portion of the annular ligament appears to be slightly infiltrated with pus cells as if there was an attempt to burst through from the labyrinth into the middle ear. *Cochlea.* Pus is present in all coils, especially in the scala tympani and the scala vestibuli (Fig. 24). In the cochlear canal itself the exudate is of a fibrinous nature. Corti's organ and the stria vascularis are desquamating. The spiral ligament is infiltrated with the pus cells (Fig. 24). (The infection appears to have passed from the internal meatus along the vessels and nerves to the labyrinth, rather than by the aqueduct of the cochlea.) There is some hemorrhage in the perilymphatic space of the vestibule around the vestibular nerve. The nerve and epithelium of the saccule and the cristae of the canals are infiltrated with pus (Fig. 26). *Internal meatus.* There is much pus within the arachnoid sheath (Fig. 24).

Deafness following Measles and Pneumonia.

The majority of cases are due to middle ear suppuration with or without labyrinthitis of middle ear origin. Siebenmann has recorded six cases of deafmutism following measles. In three the trouble was undoubtedly of middle ear origin; in the remaining three the route of labyrinth infection was an open question. In 50 to 60 per cent of cases of deafmutism following measles the tympanic membrane is normal. Meningitis symptoms in measles are not very rare and are usually attributed to the high fever.

Meningitic Neuro-labyrinthitis following Measles and Pneumonia. E. B., male, aged 17 months, admitted April 23, 1914, with shortness of breath of fourteen days' duration. The child had had measles four months before admission, followed by broncho-pneumonia. On admission, pulse 162, temperature 101.8° F. The evening temperature remained elevated. A fortnight after admission twitching of the whole of the right side developed. Lumbar puncture evacuated turbid fluid under greatly increased tension. *Post-mortem.* The right lung showed congestion and edema, with em-

physema. The left lung was collapsed and also showed bronchiectasis. There was copious pale greenish purulent exudate in the subarachnoid space. Examination of the fluid of the bronchi and from the meninges gave a pure growth of *pneumococcus.*

Microscopic examination of the Right Ear. There is slight swelling of the mucosa and a little mucopurulent exudate in the sinus tympani and in the niches of the oval (Fig. 27 and 30) and round windows.

Cochlea: There is much pus in the scala tympani in all coils (Fig. 27, 28 and 29). This extends up to the helicotrema (Fig. 28). In the scala vestibuli the purulent exudate is not so marked. There is some curdled lymph with a few pus cells in the scala media (Fig. 29). Corti's organ is well preserved (Fig. 29). The cochlear aqueduct contains pus throughout. There is purulent infiltration in the perilymph space of the vestibule (Fig. 27). (As one would expect from the route of infection, there is more pus in the cochlea than in the vestibule and canals.) *Internal meatus.* Both branches of the eighth nerve have unfortunately been torn out at the post-mortem. The fundus of the meatus shows pus. (Fig. 27 and 28).

Conclusions: (1) Meningitic neuro-labyrinthitis is a frequent cause of deafness and deafmutism.

(2) Deafness due to epidemic cerebro-spinal meningitis is certainly due to meningitic neuro-labyrinthitis. Measles and pneumonia may also be followed by meningitis, and secondary neuro-labyrinthitis. In acquired syphilis and mumps lepto-meningitis is of common occurrence and is associated with inner ear deafness, which is probably to be explained by neuritis or neuro-labyrinthitis (no microscopic examination as yet). Certain cases of deafness after influenza and osteomyelitis may also be of meningitic origin.

(3) The original source of infection may be in the respiratory tract, parotid gland, genital organs, long bones or elsewhere.

(4) A blood infection (septicaemia) probably in all cases forms the connecting link between the primary disease and the onset of meningitis.

(5) Meningitic neuro-labyrinthitis is usually but by no means always bilateral. The onset is usually sudden. Irritative symptoms, such as tinnitus and giddiness, are often present but may not be observed owing to the mental condition (coma) of the patient. In epidemic cerebro-spinal meningitis and parotitis, deafness usually occurs early in the course of the disease.

(6) Deafness due to meningitic neuro-labyrinthitis may be associated with other metastatic lesions, e. g., orchitis, arthritis, mastitis, blindness or paralysis of the oculo-motor nerves.

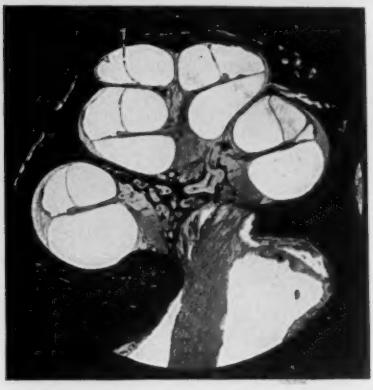

Fig. 1.

Fig. 1.—Case I. Horizontal section through left ear. No. 250. x 11 diam. Axial section through cochlea. Shows dilation of cochlear duct in all coils. The cochlear nerve and spiral ganglion appear normal. 1. Reissner's membrane is ruptured at the apex of the cochlea (artefact?), and here the basilar membrane lies on the bony lamella which separates the upper part of the middle coil from the apical coil. The helicotrema is absent.

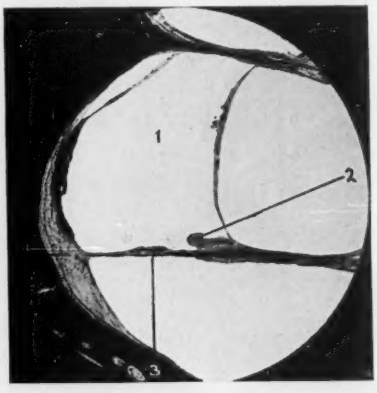

Fig. 2.

Fig. 2.—Case I. Horizontal section through lower part of left middle coil. No. 250. x 45 diam. 1. Dilated cochlear duct. 2. Membrana tectoria lying on the apex of the limbus. 3. Rudimentary Corti's organ. 4. Spiral ligament showing dropsical degeneration. The stria vascularis appears to be well formed.

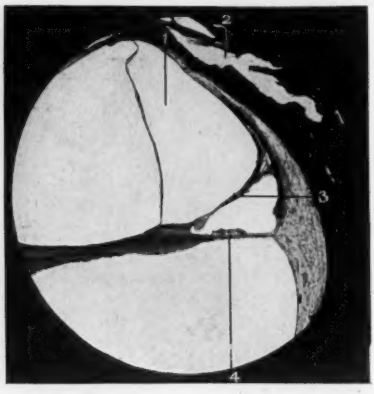

Fig. 3.

Fig. 3.—Case I. Horizontal section through upper part of left basal coil. No. 250. x 45 diam. 1. Dilated cochlear duct. 2. Split in the bony capsule (artefact). 3. Adhesion between membrana tectoria and stria vascularis. 4. Rudimentary Corti's organ.

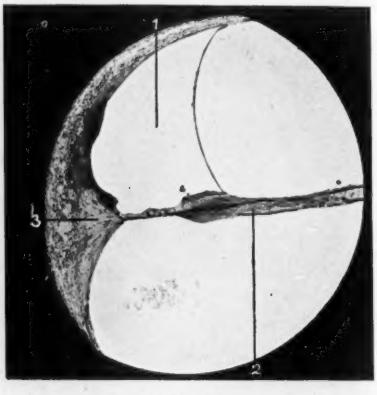

Fig. 4.

Fig. 4.—Case I. Horizontal section through lower part of left basal coil. No. 250. x 45 diam. Note that the membrana tectoria is tucked into the internal spiral sulcus. Corti's organ is rudimentary, but the inner pillar cell can be made out. 1. Dilated cochlear duct. 2. Nerve in bony spiral lamina, apparently normal. 3. Spiral ligament which shows the so-called dropsical degeneration.

Fig. 5.

Fig. 5.—Case I. Horizontal section through left ear. No. 290. x 11 diam. 1. Footplate of stapes. 2. Purulent exudate in hollow of stapes. 3. Opening of smooth end of lateral canal into vestibule. 4. Crus commune. 5. Utricle. 6. Ductus perilymphaticus. 7. Nerve to saccule surrounded by meningitis. 8. Collapsed saccule.

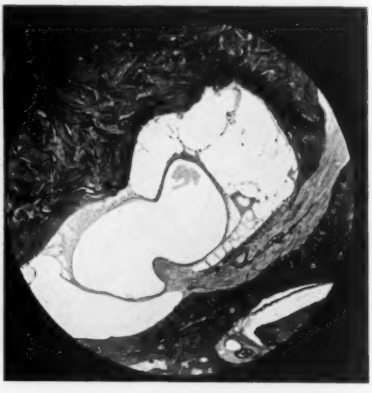

Fig. 6.

Fig. 6.—Case I. K. M'D., male, aged 13 years. Congenital Deaf-Mute. Section No. 165. Horizontal section through left ear. x 13 diam. Shows normal condition of crista of lateral canal.

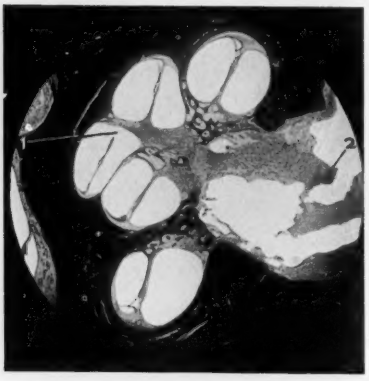

Fig. 7.

Fig. 7.—Case I. Vertical section through right ear. No. 55. Axial section through cochlea. x 8 diam. Cochlear nerve and ganglion are normal. 1. Helicotrema. 2. Meningitis infiltration in internal meatus.

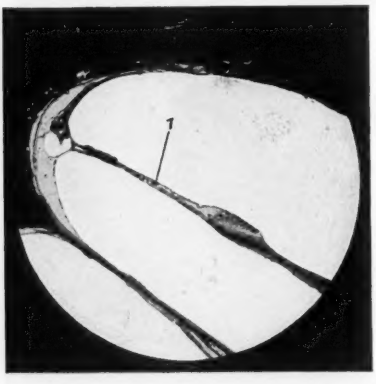

Fig. 8.

Fig. 8.—Case I. Vertical section through right ear. No. 55. x 50 diam. 1. Upper blind end of cochlear duct, showing complete collapse.

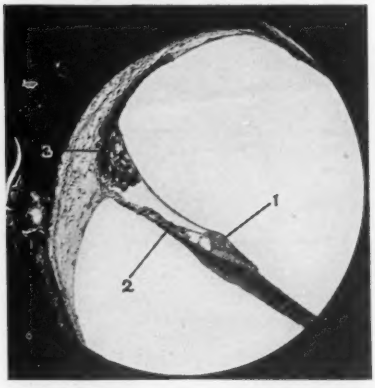

Fig. 9.

Fig. 9.—Case I. Vertical section through lower part of right middle coil. No. 55. x 50 diam. 1. Membrana tectoria, which lies between the origin of Reissner's membrane and the limbus. 2. Rudimentary Corti's organ. 3. Great proliferation of stria vascularis, which occupies the outer third of the narrow cochlear canal.

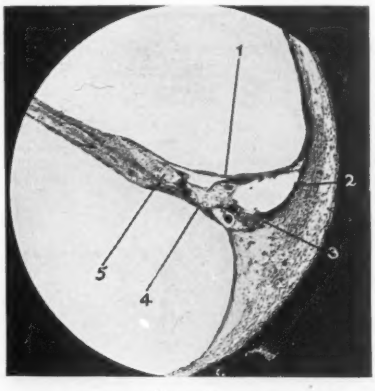

Fig. 10.

Fig. 10.—Case I. Vertical section through upper part of right basal coil. No. 55. x 50 diam. 1. Reissner's membrane depressed and attached to basilar membrane. 2. Position of stria vascularis, which is absent. 3. Position of spiral prominence. 4. Basilar membrane. 5. Membrana tectoria tucked into internal spiral sulcus. It will be seen that there is a complete malformation of the epithelium lining the cochlear canal, which is represented by at least four spaces in the section.

Fig. 11.

Fig. 11—Case I. Vertical section of right ear. No. 226. x 13 diam. 1. Collapsed saccule. 2. Nerve to ampulla of posterior canal. 3. Opening of aqueduct of cochlea. 4. Niche of round window. 5. Cochlear duct.

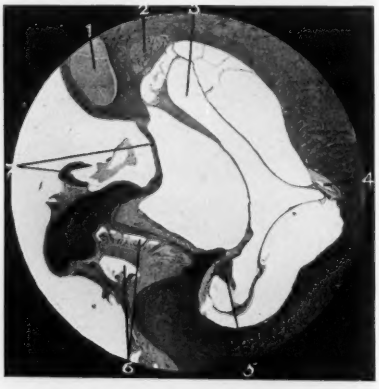

Fig. 12.

Fig. 12.—Case I. Vertical section through right ear. No. 315. x 8 diam. 1. Facial nerve. 2. Vestibular nerve. 3. Utricle (neuro-epithelium normal). 4. Endolymphatic duct. 5. Crista of posterior canal, normal. 6. Niche of round window, and, above this, the blind vestibular end of the cochlear duct. 7. Stapes. The head is lightly attached to promontory.

Fig. 13.

Fig. 13.—Case II. Horizontal section through right ear. No. 204. x 5 diam. 1. Cholesteatoma on inner wall of tympanum. 2. Promontory. 3. Tubal part of tympanic cavity. 4. Internal carotid artery. 5. Upper part of basal coil largely filled up by new bone formation. 6. Internal meatus with nerves. 7. Crus commune. 8. Smooth end of lateral canal filled with connective tissue. 9. Facial nerve.

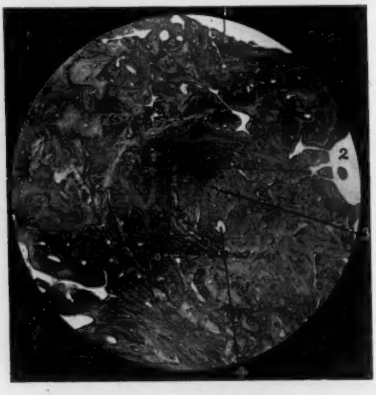

Fig. 14.

Fig. 14.—Case II. C. S., male, aged 10 years. Acquired deaf-mutism due to labyrinthitis. Horizontal section through right (operated) ear. No. 70. x 8 diam. 1. Inner wall of aditus. 2. Mastoid antrum. 3. Convexity of lateral canal almost entirely obliterated by new bone. 4. Remains of endolymphatic space.

Fig. 15.

Fig. 15.—Case II. Horizontal section through right ear. No. 105. x 11 diam. 1. Mucosa covering prominence of lateral canal. 2. Lateral canal completely obliterated by bone. 3. Erosion of bone. 4. The ampullary end of superior canals partially filled up by new bone formation.

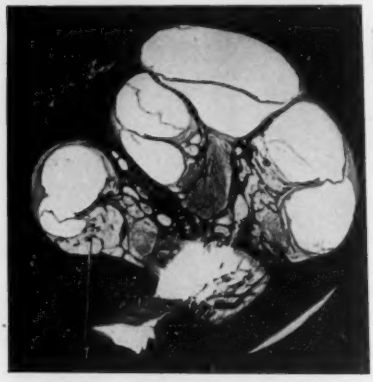

Fig. 16.

Fig 16.—Case II. Horizontal section through right ear. No. 150. x 11 diam. The cochlear duct is greatly dilated, almost obliterating the scala vestibuli. Corti's organ is completely absent. The spiral ganglion is somewhat atrophied, especially in the basal coil. 1. New formation of bone and connective tissue in scala tympani of basal coil. (The specimen has been somewhat spoilt by the evacuation apparatus.)

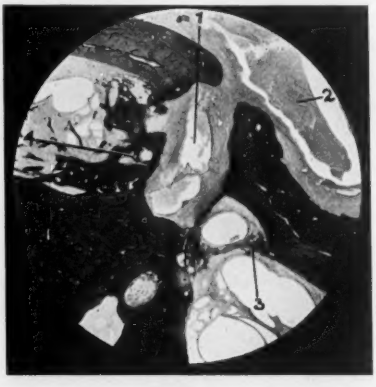

Fig. 17.

Fig. 17.—Case I. Horizontal section through right ear. No. 243. x 11 diam. 1. Niche of round window with pus cells. 2. Sinus tympani, also full of pus. 3. New bone formation in the lower part of the vestibule. 4. New bone formation in the scala tympani on inner side of the thickened round window membrane.

Fig. 18.

Fig. 18.—Case II. Horizontal section through left ear. No. 406. x 5 diam. 1. Tubal part of tympanic cavity. 2. Anterior part of tympanic membrane, with malleus. 3. Rupture of atrophic posterior portion of drumhead—artefact. 4. Facial nerve with stapedius to the left of it. 5. Pus in sinus tympani. 6. Smooth end of posterior canal completely obliterated by new bone. 7. Ampullary end of posterior canal. 8. Niche of round window. 9. Basal coil of cochlea with new bone formation in scala tympani and scala vestibuli.

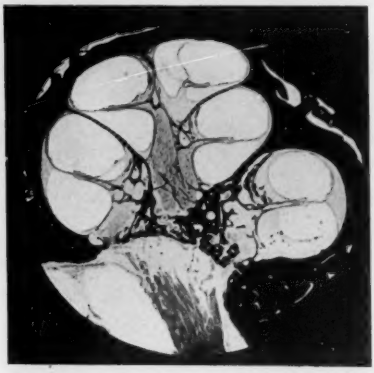

Fig. 19.

Fig. 19.—Case II. Axial section through left cochlea. No. 336. x 11 diam. Showing new formation of delicate connective tissue and bone in perilymphatic space, and great dilatation of cochlear duct.

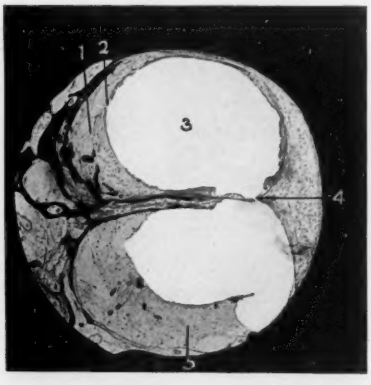

Fig. 20.

Fig. 20.—Case II. Horizontal section through lower part of basal coil, left ear. No. 336. 1. New connective tissue formation in scala vestibuli. 2. Reissner's membrane. 3. Greatly dilated cochlear duct. 4. Rupture of basilar membrane (artefact). 5. New connective tissue and bone formation in scala vestibuli.

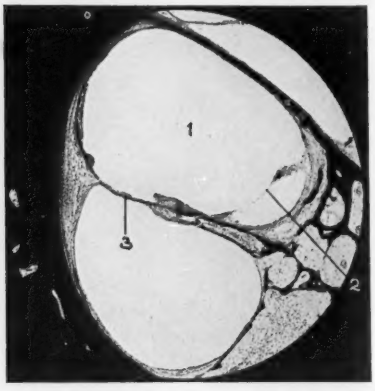

Fig. 21.

Fig. 21.—Case II. Horizontal section through upper part of left basal coil. x 30 diam. 1. Greatly dilated cochlear duct. 2. Reissner's membrane. 3. Almost complete atrophy of Corti's organ.

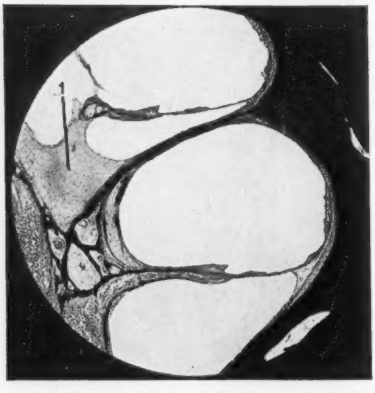

Fig. 22.

Fig. 22.—Case II. Horizontal section through middle and upper coils of left cochlea. x 30 diam. Shows dilated cochlear duct and atrophic conditions of Corti's organ. The membrana tectoria is absent. 1. New connective tissue filling up helicotrema.

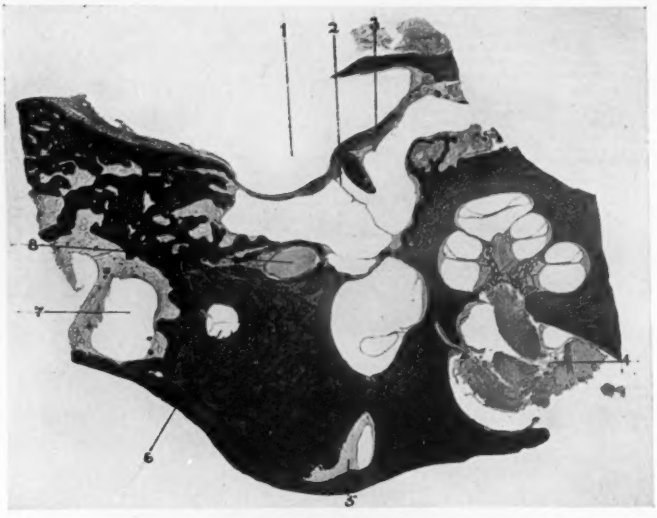

Fig. 23.

Fig. 23.—Epidemic cerebro-spinal meningitis of about eight days' duration. Horizontal section through right ear. No. 135. x 6 diam. 1. External auditory meatus. 2. Adhesion between drumhead and inner wall. 3. New bone formation in drumhead (chalk patch). 4. Internal meatus, showing slight meningeal exudate. 5. Crus commune (note faulty formation of walls of bony canal. 6. Convexity of lateral canal. 7. Antrum, with thickened lining membrane. 8. facial nerve. Note that long process of incus is absent; it should be situated between the handle of the malleus and the footplate of the stapes which is seen in the oval window. Middle ear suppuration had evidently been present in childhood in this case.

Fig. 24.

Fig. 24.—Section 70. x 9 diam. 1. Purulent exudate in helicotrema. 2. Exudate in scala vestibuli. 3. Same in scala tympani. 4. Cochlear nerve with hemorrhagic purulent exudate around. 5. Endosteum detached from wall of scala tympani of basal coil (artefact).

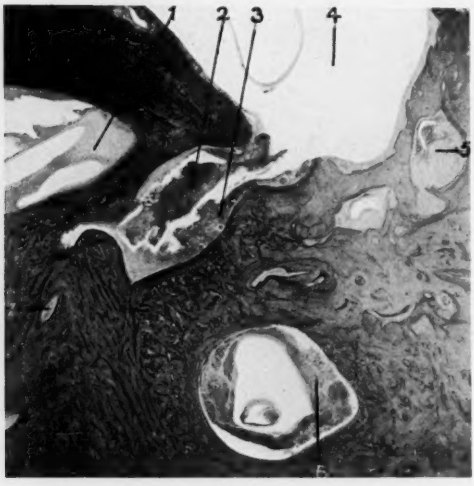

Fig. 25.

Fig. 25.—Section 205. x 9 diam. 1. Exudate in scala tympani of basal coil. 2. Purulent exudate in sinus tympani. 3. Thickened, infiltrated and engorged mucosa lining the sinus tympani. 4. Tympanic cavity. 5. Stapedius. 6. Hemorrhagic purulent exudate in perilymph space of posterior canal. 7. Aqueduct of cochlea containing exudate.

Fig. 26.

Fig. 26.—Section 50. x 9 diam. 1. Facial nerve. 2. Crista of lateral canal infiltrated with pus (the exudate is streaming through the apex of the crista, and, if the illustration is turned right round, gives the appearance of a volcano in eruption). 3. Aditus. 4. Purulent and hemorrhagic exudate in perilymph space. 5. The vestibular nerve surrounded by purulent exudate. 6. Purulent hemorrhagic exudate at point of entry of nerve to utricle.

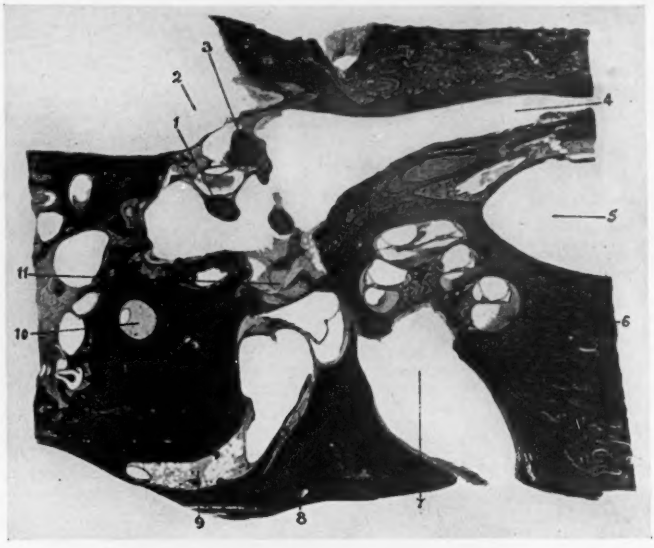

Fig. 27.

Fig. 27.—E. B., pneumonia and meningitic labyrinthitis. Section 205. x 6 diam. 1. Incus. 2. External meatus. 3. Malleus. 4. Eustachian tube, normal. 5. Carotid canal. 6. Exudate in scala tympani of basal coil. 7. Internal meatus (the nerves have unfortunately been torn out). 8. Endolymphatic aqueduct. 9. Exudate in perilymph space of crus commune. 10. Lateral canal. 11. Niche of oval window showing thickened mucosa with some exudate.

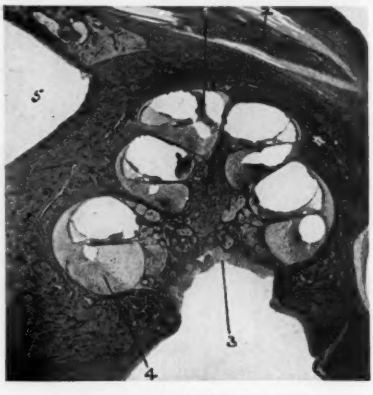

Fig. 28.

Fig. 28.—E. B., pneumonia and meningitic labyrinthitis. Section 185. x 9 diam. 1. Purulent exudate in helicotrema. 2. Tensor tympani. 3. Pus in fundus of internal meatus. 4. Purulent exudate in basal coil of cochlea. 5. Carotid canal.

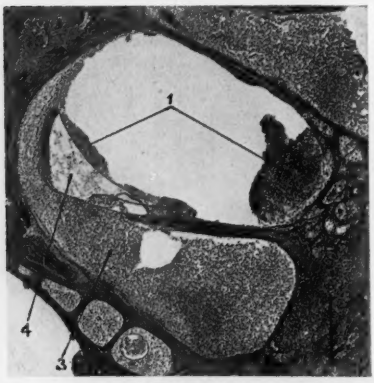

Fig. 29.

Fig. 29.—E. B., pneumonia and meningitic labyrinthitis. Section 185. x 38 diam. 1. Purulent exudate in scala vestibuli—to the left it is lying on upper surface of Reissner's membrane. 2. Spiral ganglion infiltrated with pus. 3. Scala tympani full of pus. 4. Cochlear canal with fibrinous and slightly purulent exudate.

Fig. 30.

Fig. 30.—E. B., pneumonia and meningitic labyrinthitis. Section 245. x 9 diam. 1. External meatus. 2. Facial nerve. 3. Exudate in perilymph space of smooth end of lateral canal where it joins the vestibule. 4. Exudate in niche of oval window. 5. Head of stapes. 6. Long process in incus. 7. Malleus.

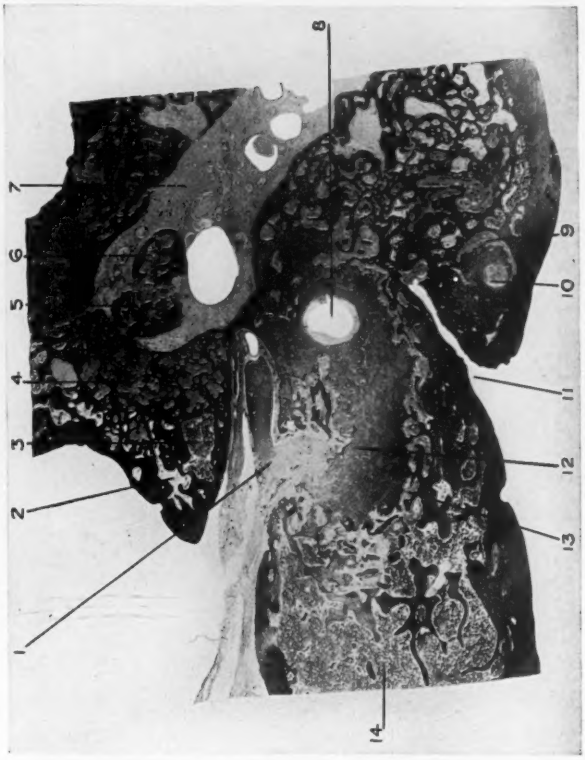

Fig. 31.

Fig. 31.—Congenital syphilitic disease of the ear. Horizontal section through left ear. No. 102. x 5 diam. 1. Geniculate bend of facial nerve. 2. Normal fatty marrow. 3. Normal cellular marrow. 4. Osteoclastic marrow. 5. Head of malleus ankylosed to attic wall; note erosions. 6. Body of incus; both ossicles show wide marrow spaces. 7. Greatly thickened submucosa of attic. 8. Ampullary end of superior canal. 9. Osteoclastic marrow eroding bony wall of canal. 10. Smooth end of superior canal filled with granulation tissue. 11. Opening of fossa subarcuata. 12. Upper part of bony cochlear capsule. 13. Osteoclastic marrow eroding cochlear capsule. 14. Normal cellular marrow in anterior part of petrous pyramid.

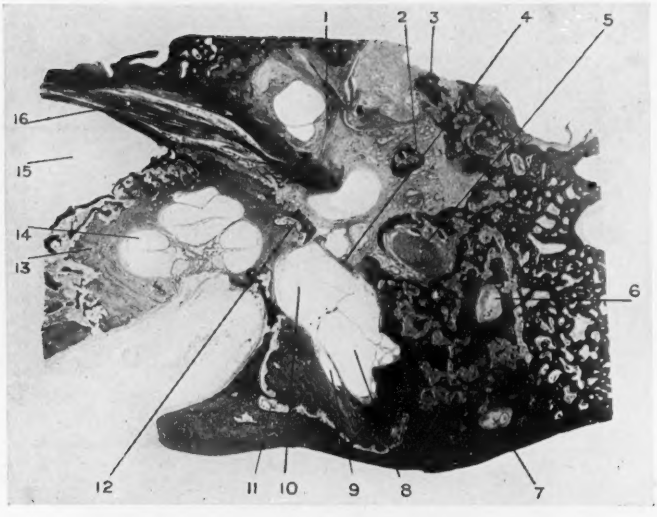

Fig. 32.

Fig. 32.—Congenital syphilitic disease of the ear. Horizontal section through left ear. No. 282. x 5 diam. 1. Tendon of tensor tympani. 2. Long process of incus. 3. Necrosed bone being extruded into external meatus. 4. Footplate of stapes. 5. Facial nerve. 6. External canal. the perilymph space is filled with granulation tissue. 7. Posterior canal: also filled with granulation tissue. 8. Utricle. 9. Ductus endolymphaticus. 10. Dilated saccule. 11. Osteoclastic marrow which, higher up, erodes the internal meatus. 12. Osteoclastic marrow in bone of promontory in anterior margin of oval window. 13. Bony capsule of cochlea. 14. Dilated scala media of basal coil of cochlea. 15. Carotid canal. 16. Tensor tympani muscle. Note the erosion of the bony capsule of the cochlea by the osteoclastic marrow.

Fig. 33.

Fig. 33.—Left ear. Section 342. x 11 diam. 1. Carotid canal. 2. Area of osteomyelitis in capsule of cochlea between cartilage and lamellar bone. 3. Dilated cochlear duct of middle coil. 4. Dilated cochlear duct of basal coil.

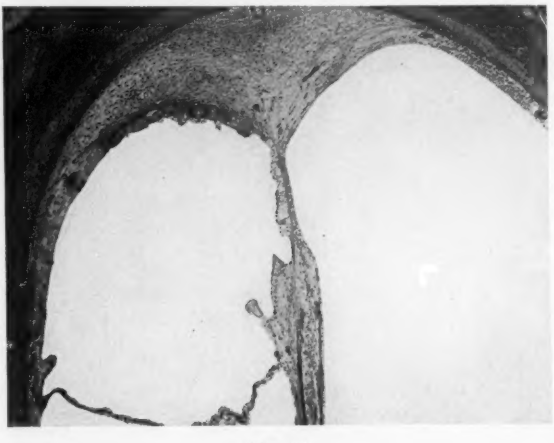

Fig. 34.

Fig. 34.—Left ear. Section 320. x 50 diam. Shows condition (degenerative neuritis) of Corti's organ in basal coil.

Fig. 35.

Fig. 35.—Left ear. Section 340. x 210 diam. (Kulschitzky stain.) 1. Vein in modiolus. 2. Spiral ganglion of basal coil, showing atrophy of nerve cells.

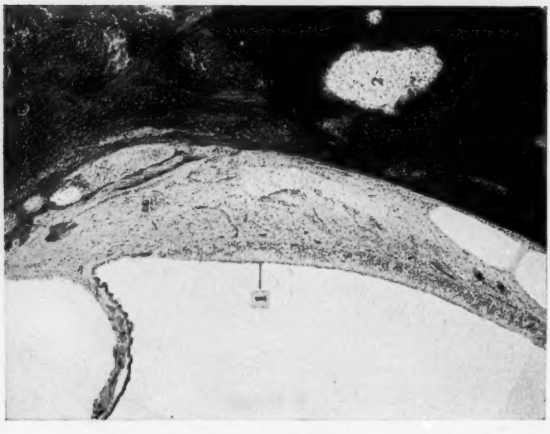

Fig. 36.

Fig. 36.—Left ear. Section 276. x 42 diam. Shows condition of, 1. Neuro-epithelium of sacculus. 2. Area of osteomyelitis in labyrinth capsule.

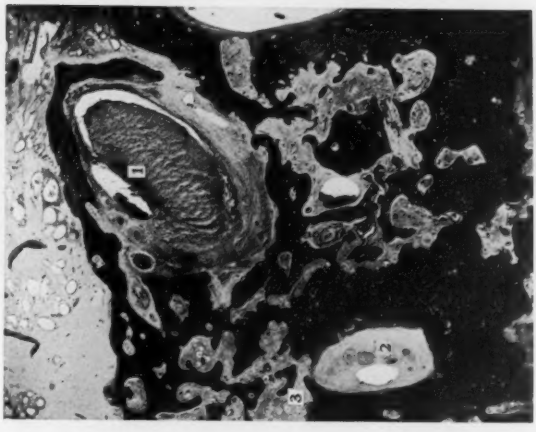

Fig. 37.

Fig. 37.—Left ear. Section 264. x 10 diam. 1. Facial nerve. 2. New connective tissue and giant cells filling perilymph space of external canal and in contact with, 3. Phagocytic marrow.

Fig. 38.

Fig. 38.—Left ear. Section 144. x 10 diam. 1. Crista of external canal. 2. New connective tissue in perilymph space in contact with, 3. Phagocytic marrow invading the labyrinth capsule. (For convenience the section has been rotated through 90 degrees.)

Fig. 39.

Fig. 39.—Section 126. x 11 diam. 1. Roof of external meatus with necrosed bone and desquamated epithelium. 2. Malleus ankylosed to attic wall. 3. Incus. Note invasion of marrow spaces from submucosa. 4. Cystic space containing mucoid exudate.

Fig. 40.

Fig. 40.—Left ear. Section 308. x 11 diam. Shows dilated and marrow parts of ductus endolymphaticus. 1.Dilated saccule. 2. Utricle. 3. Dilated part of ductus endolymph. 4. Narrow part surrounded by osteomyelitis.

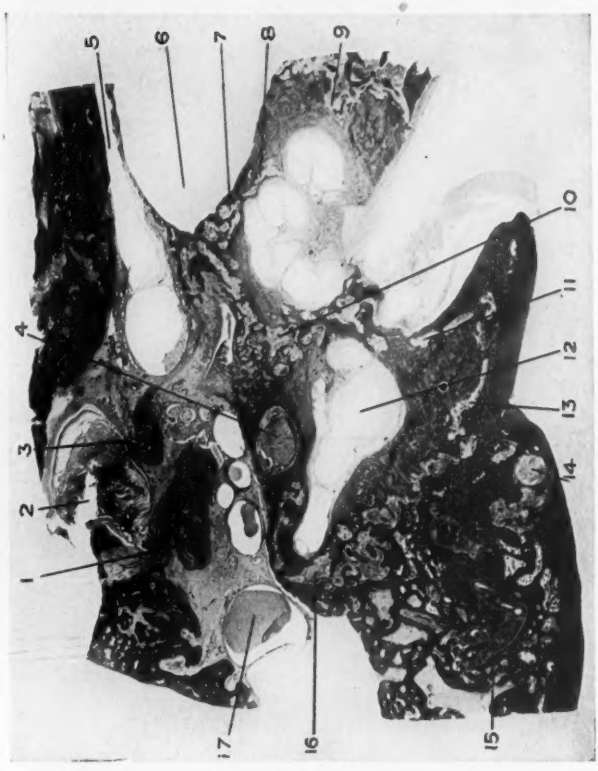

Fig. 41.

Fig. 41.—Congenital syphilitic disease of the ear. Horizontal section through right ear. No. 152. x 5 diam. 1. Incus. 2. External meatus containing desquamated epithelium and bits of necrosed bone. 3. Malleus in region of short process. 4. Facial nerve. 5. Eustachian tube. 6. Carotid canal. 7, 8 and 9. Areas of ostitis vasculosa (chronic osteomyelitis) in capsule of cochlea. 10. Ostitis vasculosa in cochlear capsule just above and in front of oval window. 11. Ostitis vasculosa near internal auditory meatus. 12. Utricle. 13. Osteoclastic marrow (ostitis vasculosa). 14. Posterior canal obliterated by granulation tissue. 15. Dilated vessels of fossa subarcuata, which here take a knee-shaped bend. 16. Ostitis vasculosa eroding bony wall of external canal. 17. Cavity of aditus containing mucoid material.

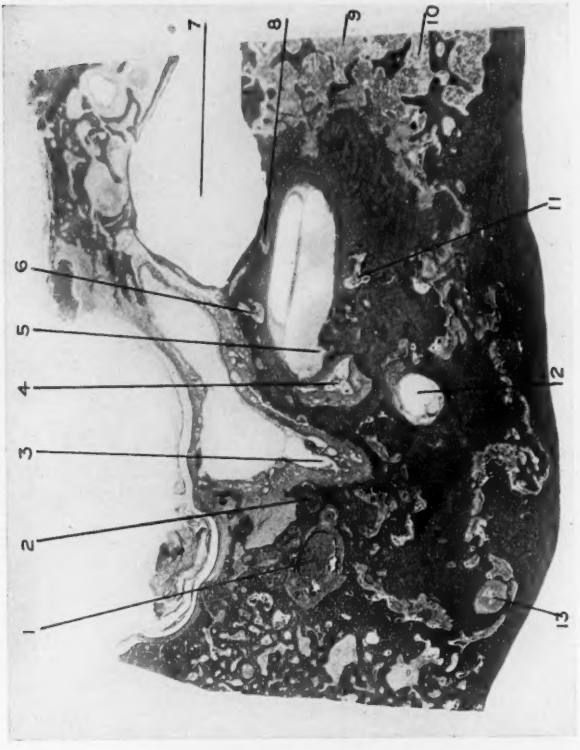

Fig. 42.

Fig. 42.—Congenital spphilitic diseases of the ear. Horizontal section through right ear. No. 274. x 5 diam. 1. Facial nerve. 2. Stapedius muscle. 3. Sinus tympani. 4. Niche of round window. 5. Cochlear opening of perilymphatic aqueduct filled up with delicate connective tissue which is only faintly seen. 6 and 8. Area of ostitis vasculosa in cochlear capsule. 7. Carotid canal. 9. Cellular marrow (normal). 10. Fatty marrow (normal). 11. Area of ostitis vasculosa behind basal coil. 12. Ampullary end of posterior canal. 13. Smooth end of posterior canal obliterated by connective tissue and surrounded by osteoclastic marrow.

Fig. 43.

Fig. 43.—Right ear. Section 165. x 7 diam. (Stained with Kulschitzky haematoxylin.) Shows cochlea and internal meatus. 1. Scala tympani of basal coil partially filled with new connective tissue. 2. Greatly dilated scala media which obliterates the scala vestibuli. 3. Area of meningitis (small cell infiltration) in internal meatus next to osteomyelitis in the bony partition, between cochlea and vestibule.

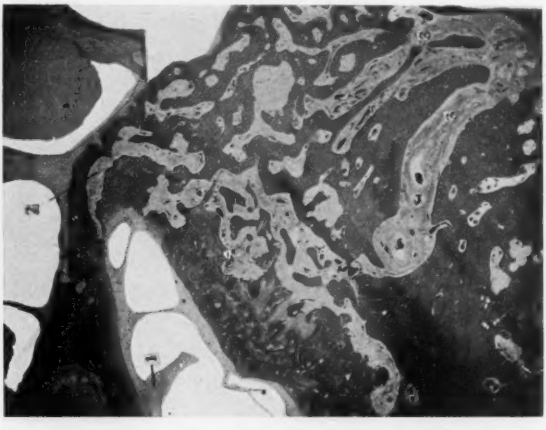

Fig. 44.

Fig. 44.—Right ear. Section 123. x 7 diam. 1. Crista of external canal. 2. Osteomyelitis reaches endosteum. 3. Vessels of fossa subarcuata.

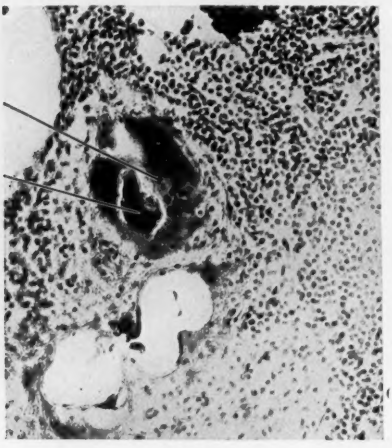

Fig. 45.

Fig. 45.—Congenital syphilitic disease of the ear. Section through osteoclastic marrow. x 200 diam. Showing large osteoclast with many nuclei; the giant cell contains a bit of necrosed bone. 1. Giant cell. 2. Necrosed bone in the giant cell.

Fig. 46.

Fig. 46.—Right ear. Section 188. x 11 diam. 1. Anterior margin of oval window with invasion of marrow spaces. 2. Footplate of stapes. 3. Dilated sacculus. 4. Dilated utriculus.

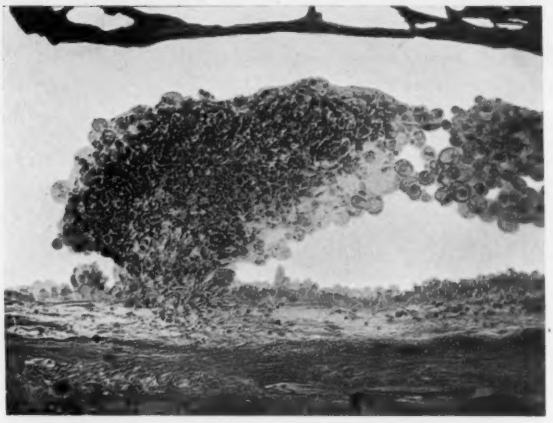

Fig. 47.

Fig. 47.—Right ear. Section 236. x 134 diam. Shows catarrhal excrescence from lining membrane of ductus endolymphaticus in the dilated portion near the sacculus.

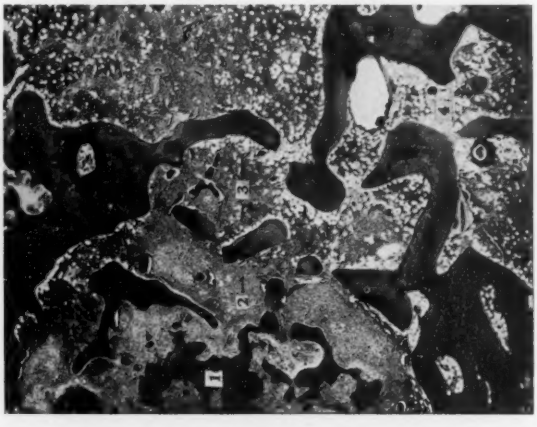

Fig. 48.

Fig. 48.—Left ear. Section 78. x 13 diam. Showing different kinds of marrow. 1. Capsule of cochlea. 2. Granulation tissue change in marrow. 3. Leucoblastic reaction. 4. Fatty marrow.

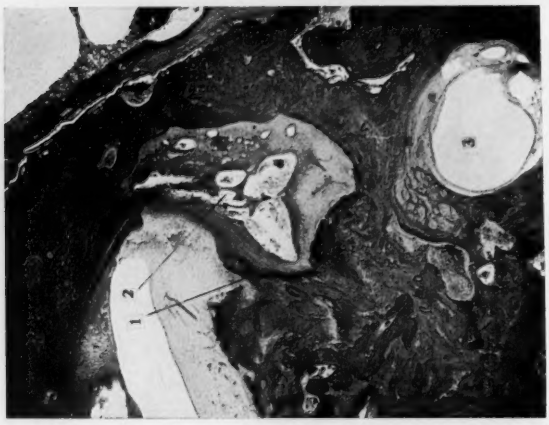

Fig. 49.

Fig. 49.—Right ear. Section 274. x 13 diam. 1. Cochlear opening of perilymphatic duct blocked by new connective tissue. 2. Niche of round window. 3. Ampullary end of posterior canal.

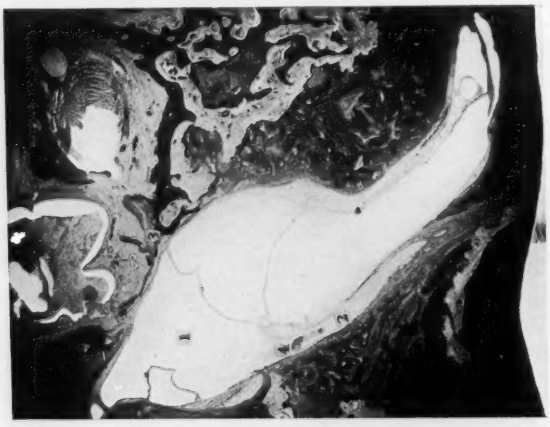

Fig. 50.

Fig. 50.—Right ear. Section 236. x 7 diam. Shows oval window, vestibule, and crus commune. 1. Dilated sacculus and ductus reuniens. 2. Dilated endolymph ductus with, 4. Catarrhal excrescence growing from inner and anterior wall of duct. 3. Stapes in oval window.

(7) The infection usually passes along the subarachnoid space from the base into the internal auditory meatus and then along the nerves and vessels to the labyrinth. In some cases the perilymphatic aqueduct is the route of invasion, while in others both paths may be involved.

(8) As a rule both the cochlear and vestibular apparatus are affected. Frequently the cochlear apparatus is the one mainly or alone involved; rarely do we have a more or less isolated affection of the vestibular apparatus.

(9) The pathological changes producing the deafness may be: (a) hydrocephalus; (b) changes in the walls of the fourth ventricle; (c) purulent infiltration of the eighth nerve with subsequent descending neuritis accompanied by atrophy of the spiral ganglion cells and Corti's organ; (d) purulent labyrinthitis which, if the patient lives long enough, is followed by the formation of granulations tissue and, later, of new connective tissue and bone in the hollow spaces of the labyrinth.

(10) The resulting deafness is as a rule complete and permanent in the ear (or ears) affected.

(11) Vestibular symptoms (loss of balancing and waddling gait) pass off rapidly in adults, but in young children they may last as long as one year.

(12) In cases of sudden nerve deafness with or without vestibular symptoms lumbar puncture should be performed and the cerebro-spinal fluid examined chemically and microscopically. The Wassermann reaction of the fluid should also be tested and cultures made.

(13) Repeated lumbar punctures are of use in treatment, especially in cases of deafness due to hydrocephalus. Small doses of potassium iodide and hypodermic injections of pilo-carpin have been used in the treatment of meningitic neuro-labyrinthitis, but apparently without success.

Intra-uterine Meningitis.

This condition is very difficult to prove. At most we can say that in cases of deafness apparently dating from birth, microscopic examination has shown the presence of those changes usually associated with meningitic labyrinthitis. Gradwohl has shown that meningitis may occur in the foetus in utero. Haike's case was one of encephalitis hemorrhagica. The infant died on the fourth day after birth. The cranial cavity contained much clear fluid, and the cerebral hemispheres were absent. There were recent hemorrhages in the eighth nerve and in all coils of the cochlea. Panse has recorded the case of a female, aged 65, said to have been born deaf. Histological

examination showed the results of an inflammatory infection of the inner ear.

Castex and Marchand have recorded three cases apparently due to intra-uterine meningitis. The middle ear was intact. There was distinct atrophy of the temporal lobes on both sides. Corti's organ, the cochlear nerve, and the central paths were all degenerated.

Alexander and Neumann have recorded the case of a female, aged 43, said to have been deaf from birth. They found the middle ear normal; cochlear and vestibular ganglia degenerated; atrophy of nerve endings of the labyrinth; dilitation of the cochlear duct; bony closure of the scala tympani at the base of the cochlea; scala vestibuli closed off from the perilymph space of the vestibule by bone.

"Congenital" Syphilitic Deafness.

Many cases of so-called "congenital" deafmutism are really due to intra-uterine syphilis or to syphilitic changes in the ear occurring before the child has learned to talk. The most severe forms of ear syphilis occur in intra-uterine life. The newly born infants show all the signs of congenital deafness and the static labyrinth is not excitable. (Alexander) In twenty-one syphilitic families Kerr Love found that there had been altogether 172 pregnancies, of which 30 resulted in miscarriages or still-births. Of the 142 children born alive, 45 had died, thus, with the miscarriages and still-births, giving a total of 75 deaths. (Among the 45 deaths which occurred after birth there were many cases of meningitis.) Of the 97 individuals living at the time of examination 31 were deaf and blind. Of the 66 living children who were neither deaf nor blind, many had been born before the parents contracted syphilis.

The percentage of cases of congenital syphilitic deafness among deafmutes varies from 3.5 to 25 per cent. The lower percentages are arrived at by clinical examination, the higher ones by the Wassermann test. The late type of congenital syphilitic deafness (Hutchinson's symptom triad) is usually an evidence of expiring syphilis. The Wassermann reaction does not discover all the cases of congenital deafness which are due to congenital syphilis. When syphilis has become latent the reaction is negative in fully 50 per cent of cases.

Pathology.

(1) *In the Fetus and in Infants.* (a) An infective process in the middle ear may involve the labyrinth by rupture of the anular ligament with consequent invasion of the vestibule through the oval window. (b) Intra-uterine meningitis in the region of the internal

meatus and neuritis (small cell infiltration) of the eighth nerve. (c) Changes in the blood-vessels have been noted by French Otologists.

(2) *In Young Children* suffering from congenital syphilis, what appears to be a case of simple Eustachian catarrh runs an unfavorable course (McBride). Treatment does no good, and the drumheads remain thickened and indrawn, while the deafness is severe and persists. We have a combination of middle ear catarrh and labyrinthine deafness. Middle ear affections in congenital syphilis show a marked tendency to break into the labyrinth.

(3) *The "Late" Type of Congenital Syphilis.* Deafness occurs (gradually or suddenly) between the seventh and thirteenth years as a rule. Interstitial keratitis is almost invariably present, while "Hutchinson" teeth are found in 50 per cent of cases. Views as to the pathology of the "late form of congenital syphilitic deafness may be summarized as follows: (a) The deafness may be due to a *neuro-labyrinthitis spreading from the meninges.* A lymphocytosis exists in the cerebro-spinal fluid in children suffering from congenital syphilis. (b) *Vascular changes* (endarteritis and hemorrhages may explain the sudden form of "late" congenital syphilitic deafness. (c) *Otitis media followed by para and peri-labyrinthitis or by invasion of the labyrinth through the windows.* The tympanic membranes are seldom normal. Out of 33 cases examined by the writer the drumheads were normal in only four instances. This points to a past attack (or attacks) of otitis media. The writer holds that, at least in some cases, the "late" form of congenital syphilitic deafness is due to syphilitic otitis media, possibly with mixed infection, which (1) invades the marrow spaces of the petrous bone and also attacks the labyrinth capsule, giving rise to chronic form of osteomyelitis, which slowly invades the perilymph space of the labyrinth (gradual onset of deafness), or (2) rapidly invades the hollow spaces of the inner ear through the oval or round windows and causes syphilitic panotitis (apoplectiform onset of deafness).

Steinbrugge has recorded a case in which the inflammatory condition had invaded the labyrinth through the oval window. In Gradenigo's case Hutchinson's symptom triad was present. Both labyrinth windows were destroyed. The vestibule and cochlea were partially filled up by new formed bone and connective tissue.

Case. Male, age 20, a blind basket-maker. The father died of aneurysm at the age of 35; the mother had four still-born children before the birth of the patient; afterwards she had three more still-born children. As a young child the patient could hear well and

spoke normally. At the age of four years he had measles followed by double otorrhoea, which persisted through life. He was slightly deaf after the onset of the middle ear trouble and the deafness gradually increased. At the age of ten interstitial keratitis was noted and six months later there was syphilitic ulceration of the pharynx, followed by contraction. At this period the deafness markedly increased, and within a year became almost complete. The patient's speech soon became very indistinct. He frequently complained of noises in the ear and giddiness, and walked in a peculiar "rolling" manner. For some time before admission the patient had been very drowsy and often went to sleep in a chair in the midst of conversation. *Examination*: The incisor teeth were notched and the cornae very cloudy. Syphilitic scars were noted round the knees. The Wassermann reaction was negative. The patient's breathing was noisy. Only fluid food could be swallowed. The nasopharynx was entirely shut off from the mouth and oropharynx by scar tissue. There was marked cicatrical narrowing of the upper aperture of the larynx, the epiglottis being adherent to the posterior wall of the pharynx. The patient became comatose and died suddenly in the afternoon following this examination. Autopsy showed syphilitic changes in the skull-cap, brain membranes, lungs, heart, aorta, and liver. The brain was markedly edematous.

Summary of the Changes found on Microscopic Examination of the Ears.

I. A chronic adhesive process in the tympanic cavity, especially in the upper parts (Fig. 31, 32, and 41). The sub-mucosa is greatly thickened, and shows cystic spaces filled with mucopus (Fig. 31 and 41), but there is no perforation of the tympanic membrane.

II. Ankylosis of the malleaus (Fig. 31) and incus on the right side) to the external attic wall.

III. Necrosis and exfoliation of bone from the posterior superior wall of the external meatus (Fig. 39 and 41).

IV. Invasion of the labyrinth capsule from the deep layer of the swollen and infiltrated submucous tissue of the attic, aditus, and antrum, and to a less extent from the sub-mucosa of the lower part of the tympanic cavity (Fig. 32, 38, and 42).

V. Marked changes in the marrow surrounding the labyrinth capsule, of the nature of a chronic osteomyelitis (Fig. 37, 44, 48, and 50). These changes in the marrow are continuous, the inflammatory process spreading in from the deep layer of the submucosa of the middle ear. The marrow around the labyrinth capsule shows marked leucoblastic reaction (Fig. 48), while that in immediate contact

with the cartilage bone of the labyrinth capsule is converted into granulation tissue (Fig. 44), and shows numerous giant cells (Fig. 45) but no caseation.

VI. Erosion of the bony capsule of the labyrinth by the phagocytic marrow to such an extent that the inflammatory changes reach the endosteum of the labyrinth, especially in the region of the three semicircular canals (Fig. 37, 38, and 44). The bone which is being eaten away stains very deeply with basic stains (Fig. 33, 48 and 50), e. g., haemalum of haematoxylin. The alteration in the staining reaction of the bone appears to be due to toxic influence. In the anterior margin of the oval window the changes in the bone resemble those seen in otosclerosis. (Fig. 43 and 46).

VII. Filling up of the perilymph space of the canals (Fig. 31, 32, 37, 38, 41 and 44), especially at their convexities by granulation tissue which contains giant cells. This process has resulted in compression or obliteration of the endolymph space in these regions (Fig. 31, 41 and 42).

VIII. Obstruction of the endolymphatic acqueduct as it passes through the bone to the posterior cranial fossa. There is a catarrhal swelling of the lining membrane of the duct (Fig. 47 and 50).

IX. Great dilatation of the membranous labyrinth, especially of the utricle, saccule, vestibular end of the endolymphatic duct (Fig. 40 and 50), and of the ductus reuniens. The basal and middle coils of the cochlea are also dilated (Fig. 33, 34 and 43).

X. Formation of new connective tissue in the scala tympani just above the round window (Fig. 42, 43 and 49), and to a less extent in the scala tympani of the basal and middle coils of the cochlea. On the right side the cochlear opening of the peri-lymphatic aqueduct appears to be blocked (Fig. 49), but on the left side it is patent.

XI. Secondary degenerative neuritis of the nerve structures of the membranous labyrinth (Fig. 33, 34 and 35). This is especially marked in the basal and apical coils of the cochlea, but less marked in the middle coil. The neuro-epithelium of the saccule, utricle, and ampullae of the canals (Fig. 36 and 38), has not suffered to the same extent as that of the cochlea. The cochlear ganglion (Fig. 35), shows atrophy of the cells in all coils, especially in the basal coil, but the vestibular ganglion shows less marked changes. The facial nerve appears healthy throughout, while any changes present in the stem of the cochlear and vestibular nerves are very slight.

XII. Slight small cell infiltration within the arachnoid sheath at the fundus of the internal auditory meatus (meningitis); this ap-

pearance is only seen in the area where the marrow changes penetrate the dural and arachnoid sheaths of the nerves.

Beck has taken a stereo-radiogram of the ear in a case of active syphilitic labyrinthitis, and found a large area of rarefaction in the labyrinth capsule. Beck held that this appearance was due to a syphilitic osteo-porosis.

Hennebert was the first to call attention to the presence of "compression" nystagmus in some cases of congenital syphilitic disease of the ear. In these cases the rotation reaction is always absent, while the caloric reaction is reduced. Alexander thinks that Hennebert's sign is due to a change in the nerve endings, while Barany holds that it is due to excessive mobility of the stapes. The findings in the case of congenital syphilitic deafness described above point to another explanation, namely, that the bony wall of the external canal may be eroded by inflammatory changes in the marrow, and that the hollow space of the membranous canal may be occluded by syphilitic granulation tissue in the perilymph space, so that the rotation reaction cannot be produced. The vertical canals may also be affected, so that the caloric reaction is feeble or absent. The more powerful pneumatic test is, however, able to bring about an abnormal (reversed) fistula symptom.

Walker Downie's classical case appears to support the view of the pathology of congenital syphilitic deafness put forward in this paper. Downie's patient became deaf at the age of eleven years, the deafness being accompanied by severe pain in both ears. Otoscopy showed the drumheads to be indrawn and opaque. The patient died from meningoencephalitis resulting from a fungating gumma of the right parietal bone. The stapes was ankylosed in the oval window. The semi-circular canals were obliterated, and the vestibule largely filled up by new bone formation. The modiolus was thickened. These findings correspond with a stage of sclerosis and ossification following an attack of panlabyrinthitis, secondary to ototis media.

The writer wishes to acknowledge his indebtedness to the Carnegie Trust for their kindness in providing the illustrations which accompany this paper.

PRESENTATION OF PATIENT IN WHOM A DIAGNOSIS HAS BEEN MADE OF PLASTIC LABYRINTHITIS, PROBABLY OF CONGENITAL LUETIC ORIGIN.*

DR. WILLIAM G. SHEMELEY, JR., Philadelphia, Pa.

The case offered for your consideration, is that of a young male, aged 18 years, who first presented himself for examination on July 15, 1922.

The following is the history as related by the patient and added to by the patient's mother:

The patient has been deaf in his right ear as long as he can remember. For the past two years he has had several abscesses in his right ear accompanied with pain. The patient is not sure as to whether the condition was one of boils or a middle ear abscess. For seven days he has had a cold in his head and in the morning he awakened with a feeling of fullness in the left ear and everything sounded far away.

The patient complains of noises in his head and at times suffers from dizziness, but the vertigo has never been severe enough to cause him to go to bed. The patient's mother added to this history the facts that when one year old the young man developed a running ear. The discharge lasted three or four days and then subsided.

The mother does not remember which ear it was that discharged. The mother has had eight children, all living and in good health. One brother had abscesses in the ears. The patient's father died suddenly at the age of 56 years, from supposed acute dilitation of the heart.

Examination, Nose: Deflection of the septum to the left, bilateral spine along the suture line. Posterior portion of the spine on the left is in contact with the inferior turbinate on the left. Left middle turbinate is visible before shrinking and is in contact with the septum and lateral wall. Right middle turbinate is visible before shrinking, in contact with the septum; small space between it and the lateral wall. Compensatory hypertrophy of the right inferior turbinate. Mucous membrane much redder than normal. Patient has acute rhinitis.

Throat: Tonsils medium size; submerged. Cheesy deposits, both sides on pressure. Secondary catarrhal pharyngitis.

*Presented at Philadelphia Laryngological Society, Oct. 7, 1922.

Functional Hearing Test:

Right Ear, not heard, Conv. Speech. Not heard, Whisp. Speech. Not heard, Akumeter.

Left Ear, Conv. Speech, 8m+. Whisp. Speech, 4m. Akumeter, 6m.

Weber: Normal; Schwabach, normal. Rinne, +30″.

Not heard, Cl., sh. 29″. Not heard, C4, normal. Not heard, air, sh. 12″.

With a 3m speaking tube in the right ear and the noise producer in the left ear, the patient could not repeat the loudest spoken words.

Otoscopic: Right Ear: Cerumen along floor of the canal obstructs view. After syringing, membrane apparently intact. There is a bony exostosis which somewhat obstructs the view. A large chalky deposit occupies the posterior quadrant. The membrane is dull, opaque, retracted. By Politzer inflation, very limited motion, but the membrane returned promptly.

Left Ear: Cerumen along the floor of the canal obstructs the view. After syringing out wax, the membrane is found intact, dull, opaque. A chalky deposit is in the upper posterior quadrant. the long process of the anvil is not visible. The hammerhandle is foreshortened, denoting retraction. Normal mobility with Seigle. By Politzer inflation, the membrane came out over a fair sized area and returned promptly.

Examination for Spontaneous Nystagmus July 22, 1922: When looking to the extreme left, patient has a mixed horizontal and rotary nystagmus to the left.

When looking to the extreme right, patient has a mixed horizontal and rotary nystagmus to the right, which appears to be slightly greater than that, when looking to the extreme left.

When looking straight ahead with eyes fixed on a distant object, the patient has a very fine spontaneous rythmic, rotary nystagmus to the left.

Fistula Test: Negative to compression and aspiration, both sides.

Galvanic Test:

Right Ear: Kathode, 2 ma., increased nystagmus; Anode, 5 ma.

Left Ear: Kathode, 5 ma., increased nystagmus; Anode, 2 ma.

Turning Test, Head Erect:

10 turns to the left, 10″; 10 turns to the left, 10″; 10 turns to the right, 10″; 10 turns to the right, 8″.

Head Forward, 90°:

10 turns to the left, 10″; 10 turns to the right, 13″.

Comment: Excursions of nystagmus were coarse and tended to cease abruptly.

Head Erect: 10 turns to the right, patient stood erect without hesitating and did not tend to fall in any direction. Feet together.

10 turns to the left, patient stood erect without hesitating and did not tend to fall in any direction. Feet together.

Caloric Test, Head Erect: After syringing the right ear with cold water for 3 minutes, a rotary, rythmic nystagmus to the left (opposite side) was obtained. Inclining the head to the left shoulder changed it to horizontal nystagmus to the right (same side).

After syringing the left ear with cold water for 2 minutes, a rythmic, rotary nystagmus to the right was obtained. Inclining the head to the right shoulder changed the nystagmus to the horizontal type, to the same side. The effects were neutralized with hot water.

Examination for Spontaneous Nystagmus: July 29, 1922. On looking straight ahead there is a fine rythmic, rotary nystagmus to the left.

Turning Tests, Head Erect:

Right Ear, 20 turns to the left, 9″.

20 turns to the right, 13″.

Comment: Turning to the left, head erect, the after-turning nystagmus had some rotary element present.

Head Forward, 90°:

20 turns to the left, 13″; 20 turns to the right, 12″.

The excursions were wide and tended to cease abruptly.

Examination for Spontaneous Nystagmus: August 5, 1922. When looking straight ahead at a distant object, there is a fine, slow, mixed horizontal and rotary, rythmic nystagmus to the left. (3 in 5 seconds.)

Turning Test, Head Erect:

Right Ear, 30 turns to the left, 11″. Left Ear, 30 turns to the right, 12″.

Eye Examination: Vision O. D., 6/4½ scant. O. S., 6/4½.

External examination revealed nothing pathologic. Ophthalmoscopic examination revealed the fact that each fundus was normal.

Summary: The patient although he had been treated previously in several clinics was never subjected to an examination of the static labyrinth. His acoustic function had been tested only in a very superficial manner.

Analysis of the functional hearing tests shows the cochlear branch of the VIII nerves of the right side to be out of function, in that

with the 3 meter speaking tube in the right ear and the noise producer in the left ear, the patient could not repeat the loudest spoken words.

Since the otoscopic examination was made the patient has had discharge from the right ear. Inflation after the method of Politzer, revealed a small perferation in the region of the eustachian tube.

The galvanic test shows the nerve to still be reactive.

The turning tests show the function of the static labyrinth of the right side almost completely out of function. The figures for the right side are indicative of those found in cases that have had sufficient time to readjust themselves to the loss of labryinthine function on one side.

The caloric test proves that there is still some function remaining on the right side.

This patient has remaining, good function in the left ear. If the probably causative factor of the condition in the right ear could be found and removed or treated the possibility of loss of function on the left side would be greatly lessened.

Recalling that the patient's father died suddenly of an obscure heart disease, while comparatively a young man and that an unrecognized or improperly treated lues is often the etiological factor in such heart cases, and knowing that syphilis is prone to cause plastic labyrinthitis, prompted the taking of a specimen of the patient's blood for serological studies. The Philadelphia Clinical Laboratory reported the case as positive to all antigens.

The condition, therefore, was diagnosed as a case of plastic labyrinthitis of the right ear, probably of congenital luetic origin. The patient was placed on active antiluetic treatment. The correction of the obstructive nasal condition and the removal of the diseased tonsils has been recommended to lessen the liklihood of the onset of catarrhal middle ear conditions in the left ear.

1724 Spruce Street.

SURGICAL EMPHYSEMA FOLLOWING TONSILLECTOMY.†

DR. SIMON STEIN, New York.

Surgical emphysema per se is not a very usual occurrence. On looking through the literature I can find no report of this condition occurring during or immediately following tonsillectomy and so wish to place this case on record.

Case: H. G., boy, seven years of age, referred to me by Dr. Michael Jaffre for the removal of tonsils and adenoids.

In general appearance this boy resembled the usual child of that age that has been handicapped by hypertrophied diseased tonsils and adenoids. He was a thin, sallow, anemic looking child, weighing about fifty-five pounds. Coagulation and bleeding times were normal.

He was placed on the operating table and anesthetized, using straight ether with the open mask. When sufficiently under, a mouth gag was inserted and the anesthesia was continued with the ordinary suction pressure apparatus.

First the left tonsil was removed, using the sluder instrument with a dulled blade. There was no adhesions present, the tonsil shelled out easily, and was separated away from the posterior pillar by the tip of my index finger. The bleeding was very slight and no sponges were used. The other tonsil was then removed in the same way, and here also there was very little bleeding, so that just a little suction cleared the operating field. At this time, the child gagged a little, being only slightly under anesthesia.

I turned to pick up an adenotome, and was startled by a sharp exclamation from the anesthetist. Looking around quickly, I received quite a shock. What had been a thin sallow face was now bloated up to about twice its size, the eyes closed over by tremendous bulging eyelids, the scalp lifted up high, the cheeks ballooned out, the lips puffed up and cyanotic, and respiration seemed to have ceased. I placed my hand on the face, and felt a parchment-like crackling, which I immediately identified as

†Read at New York Academy of Medicine, Section on Laryngology and Rhinology, Jan. 24, 1923.

From the Otolaryngological Department, Beth Israel Hospital, New York City.

emphysema. The child started breathing evenly, and I then examined him with a little more ease of mind.

The tissues of the face, neck, scalp and down on the chest anteriorally and posteriorally to the level of about the sixth rib were infiltrated with air. The scalp was completely lifted away from the cranium. The picture presented was as if one had inserted a tube into the superficial areolar tissues and pumped air into them, the skin rising as if made of rubber. The infiltration was entirely superficial, not involving the larynx or trachea. It seemed to have come on within a fraction of a second, coming up from the neck and extending rapidly over the face and scalp.

The child was breathing without any effort, there was no bleeding and so no further manipulation was attempted, the adenoid tissue being left in situ. There were no complications, the child being kept in bed for two days, under the careful observation of Dr. Jaffre. The emphysema went down very rapid, ly, first disappearing around the eyelids and cheeks, and then gradually from the scalp and neck, so that by the end of a week, there was only a little crackling apparent over the anterior and posterior aspects of the chest. The child at this time was playing around the house, perfectly normal, showing absolutely no ill effects from his unusual experience.

Comment: Surgical emphysema occurs as a not unusual complication of either chest surgery or injury. This may occur as a result of puncture of the pleura or lung, by surgical instruments, as in tapping of the chest, or by broken ribs or foreign bodies. Air escapes into the superficial areolar structures and diffuses rapidly, aided as a rule by the pressure from the expanding lung. This same condition can be duplicated anywhere in the body, where there are air containing structures which are punctured, and the contained air escapes into surrounding tissues of an areolar structure, which allows rapid diffusion. Such a condition will occur for instance in opening up the abdomen, or in puncturing one of the viscera, like the stomach. In the region of the nose and throat, we have a large area in which emphysema may and does occur. It may be caused by breaking into any of the accessory nasal sinuses, as in the case of Morgan[1] who reported a case of "fracture of the frontal bone into the frontal sinus with air extravasation into the cellular membranes of the eyelids." There are the cases following ethmoid operations where the patient forcibly blows the nose immediately after the operation and drives the air into the tissues.

There are also the emphysemas produced in puncturing the nasal antrae, where air is forced in first so as to expel the antral contents, and the opening of the trocar being wholly or in part in the tissues instead of the bony cavity, the air spreads in the tissues. A not unusual occurrence is one produced during the process of inflating the middle ear, where an abrasion is caused in or about the eustachean tube during the manipulation, and the air is forced into the tissues. Ehrlich[2] reports a case occurring during an intratracheal anesthesia, for a kidney operation, in which on opening the abdomen, the retro-peritoneal tissues were found infiltrated with air. Three quarters of an hour later, the operation having continued during this time, the emphysema appeared suddenly over the face and neck. Luke[3] also reports a case, almost similar to that of Ehrlich but in his case it appears that a long tube was used for the anesthesia and here a bronchus was distended by the pressure and ruptured, with the resultant emphysema.

Conclusion: It is rather interesting to theorize as to the why and wherefore of this occurrence, and it may not be amiss to refer to some of the important anatomical conditions present, which render this complication possible.

The bed of the tonsil is made up essentially by the superior constrictor muscle of the pharynx. In the child this muscle consists of but a few fibers. Outside of this is the buccinator fascia, which loosely invests the constrictors of the pharynx and extends up to the buccinator muscle. This also is very thin and in parts defective. Beyond this fascia layer we have a large cavity, containing the great vessels, loosely invested by areolar tissue which extends superficially over the head and neck and chest and allows easy egress and chance for diffusion of air. Occasionally on removing a tonsil in a child, this areolar tissue will be seen projecting out through a cleft in the muscle bed.

With such an anatomical picture in mind, there appears two ways in which this condition may have occurred.

1. When the child gagged as a result of the light anesthesia, there was the production of a sudden increase of pressure in the mouth, which at that moment was more or less of a closed cavity, as a result of the following factors, (a) large amounts of adenoid tissue in the nasal pharynx, (b) my hands and instruments at the oral orifice, (c) closing off of tht larynx by the gagging. This pressure forced the air in the mouth through an opening in the muscle beds of one or the other tonsil areas. The opening in the muscle bed, being either an anatomical de-

fect, or a result of a tear, during the separation of the tonsil.

2. This may be more likely. The ether and air mixture from my pump apparatus was forced through this same tonsillar bed at the time when the more or less closed cavity was produced as stated. Certainly the point of entrance must have been in the tonsillar beds, as there was absolutely no trauma to the wall of the pharynx, no instruments or sponge being used other than the sluder.

This case is rather unique, and is well worth remembering as having occurred and liable to occur again.

REFERENCES.

1. MORGAN: *Lancet*, 10:3:3, 1826.
2. EHRLICH, S. D.: *N. Y. Medical Record*, Nov. 28, 1914.
3. LUKE: Surg. Gyn. & Obstetrics, 1913, Vol. XVI, p. 204.
4. DISCHLER: Aus der Koeniglicher Frauenklinik in Dresden, 1898.
5. MELTZER: *J. A. M. A.*, May 16, 1914.
6. ALEXANDER: *J. A. M. A.*, March 29, 1911.

306 East 15 Street.

TRACHEOTOMY; IMPROVED TECHNIQUE.

DR. ELBYRNE G. GILL, Roanoke, Virginia.

There are times when all laryngologists are called upon to perform a tracheotomy in cases when intubation will not answer:

a. When intubation tubes are not available or their use is not understood.

b. In excessive edema of the larynx when the intubation tube does not relieve.

c. When the membrane is in the lower tracheal tract.

The accidents which may be incident to this operation are:

a. Failure to open the trachea, especially in very fat children.

b. Severe hemorrhage when the incision is carried too far to either side.

c. Irregular incision making it difficult to introduce cannula.

d. Asphyxiation from blood escaping into the trachea.

e. Asphyxiation from collapse of the trachea.

f. Septic pneumonia or lung abscess caused by the escape of blood and infected material into the trachea and bronchi.

The above complications can be avoided, also the haste in performing the operation can be obviated by employing the following technique:

Before performing the operation introduce the bronchoscope and allow it to remain in the trachea and bronchus until the operation has been completed.

All that is necessary in employing this technique is to be equipped with one bronchoscope, one laryngoscope and a pocket battery, with its connections. A competent laryngologist should be able to expose the larynx and introduce the bronchoscope in one minute's time.

When the bronchoscope is once introduced there will be no need for undue haste in performing the tracheotomy as the patient will have been provided ample breathing space and the operator will have a firm land mark to follow in cutting down on the tracheal rings. After the incision has been made through the tracheal rings and all bleeding controlled, the operator can then remove the bronchoscope to the point of the incision and then introduce the tracheal cannula and this completes the operation.

The incision should be made low, following the method described by Jackson. If this is done a tracheotomy will be performed and not a laryngotomy.

As far as the writer has been able to learn this technique is not being employed and has not been mentioned in any recent text book on laryngology.

612 MacBain Building.

AN INTERESTING CASE OF LARYNGEAL STENOSIS.*

DR. E. M. JOSEPHSON, New York.

The patient, a laborer about 50 years of age, was admitted on May 15, 1922, with the chief complaints of hoarseness, dyspnea, and a choking sensation in the throat. The onset had been acute, eight days before admission, with chill, fever, coughing and shortness of breath. Though there had been a slight improvement in the first two days of illness, on the third day the patient's condition suddenly grew worse, and, in addition to his previous complaints, he had pain in his neck and blood-tinged sputum. On admission, examination yielded the following:

Previous History. The patient had been under treatment for lues several years previously, but date of infection was not recalled. Otherwise negative.

Physical Examination. The patient appeared seriously ill; was dyspneic, cyanosed, and practically aphonic. Temperature was 38½ C. His tongue was coated and pharyngeal mucosa, reddened and granular; stomatitis, the type of which is not mentioned. External examination of neck was negative. Chest showed dullness over right lung and increased fremitus; breathing was bronchial, most markedly over middle lobe. Heart was negative; hypertension. Laryngoscopic examination revealed a laryngitis. The right cord was markedly infiltrated, especially in the posterior half. On May 20, a subsidence of the process was noted, but the infiltration in the right cord was still present. X-ray of the lung was negative. The Wassermann reaction was ++++. The examination was otherwise negative.

The patient was placed on mixed and salvarsan treatment for a period of approximately a month, but his condition remained unchanged. On June 13, tracheotomy was performed, in order to relieve the increased dyspnea. After tracheotomy, the pulmonary signs cleared up promptly. Mixed treatment was then resumed. About this time laryngoscopy showed a tumor bulging and displacing the right cord, covered by a smooth, healthy mucous membrane, and obstructing the glottis practically completely. The right arytenoid cartilage was immobile. Tissue removed for biopsy was returned with the diagnosis "normal mucous membrane". A tentative diagnosis of "perichondritis" was made.

*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, January 24, 1923.

On August 9, 1922, I performed a laryngofissure with the object of removing the affected cartilage. The tracheotomy wound was extended and the larynx fissured from below upward. In the right half of the larynx there was found a large, dense, sharply demarcated tumor, which displaced the true and the false cords, obliterated by pressure the sinus morgagni and filled the larynx. The mucous membrane, over the tumor, was normal in appearance. Opposite the posterior pole of the tumor, there was markedly developed a structure which has recently been described by me.

This structure consists of a sinus on the inner laryngeal wall, bordered by prominent lips, located below the true vocal cords, and

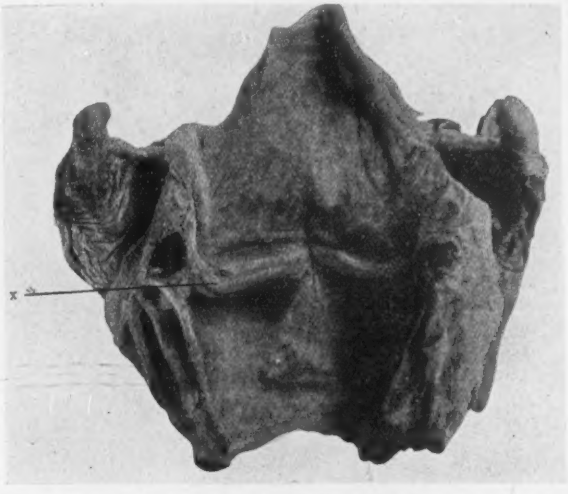

Fig. 1. Larynx showing at (x) organ in question. (2) is a prominent well-developed laryngeal tonsil attached to the aryepiglottic fold.

below the crico-arytenoid joint. Underlying this sinus is a group of glands, drained by a common duct opening into the sinus. In the average normal case this structure is recognizable only as a thickening of the mucous membrane at the point mentioned in the center of which a depression may be noted. In many normal cases this structure assumes a marked prominence (Fig. I). Histologic sections reveal the picture illustrated in Figure II, a cross-section of a normal larynx, in which we find a sinus bordered by prominent lips, into which there opens a duct (in this case about 2 mm. in diameter), draining a group of glands which lie between the mucosa and M. Vocalis. The swellings constituted by the

glands form the lips of the sinus The gland may also extend upward into the vocal cord. It resembles the sublingual and submaxillary glands in that it is composed of both tubular (mucus) and acinar (serous) alveoli, contained within one capsule (Fig. III), all of which apparently drain into a common duct. It might be mentioned, in passing, that Maziarski noted many years ago the peculiar compound tubular structure of some of the glands of the larynx. Although various groupings of the secretory glands in the larynx have been described, no mention has been found of a gland group identical in position and character of composition with the one here described, and similarly drained by a common duct.

Fig. 2. Cross section of the structure illustrated at (x) in Fig. 1. (1) is sinus, (2) common duct draining glands, (3) (4) vocal cord, (5) Sinus Morgagni.

The tumor found by me at operation in our case bore macroscopically the appearance of a pathological change in the above-mentioned structure. The fissured cricoid and thyroid cartilages were found to be normal. Inasmuch as the mucosa over the tumor was absolutely intact, a submucus enucleation of the tumor was undertaken. The planes of cleavage were well defined, and the tumor readily shelled out. At the bottom of the wound there lay free the arytenoid cartilage. Because it showed signs of dry gangrene, to all appearances due to cutting off of blood supply by the

tumor, it was removed. The vessels were ligated, the tracheal canula replaced and the laryngeal wound packed and drained.

The patient made an uneventful recovery, excepting the mild symptoms caused in the first few days after the operation by post-operative edema. When examination on October 8 revealed that the wound was completely closed, dilatation by means of the Thost bougies and Schrotter tubes was resorted to. The patient was now once again able to breath comfortably through his larynx, which showed a fairly large and free glottic space.

The pathologic specimens (Fig. IV) are of interest. The tumor (1) was about 1½x1x0.5 cm. in size, and dense and fibrous in consistency. The arytenoid cartilage (2) was gray in color and,

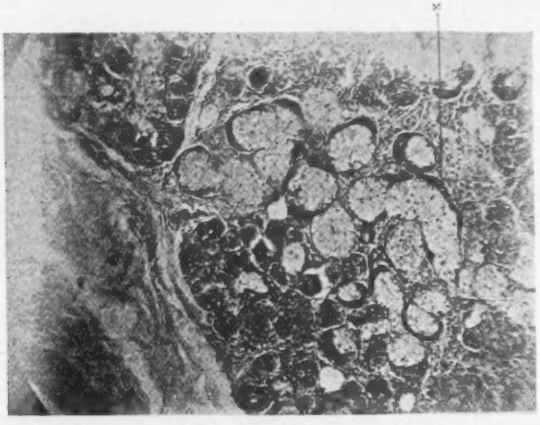

Fig. 3. Glands (3) of Fig. 2 magnified 100x. Tubular nature is seen at (x).

though intact in form, showed a dry gangrene and was putrid. The dry gangrene which it had undergone was unquestionably due to the cutting off of its blood supply by the neighboring tumor; for had the process originated as a primary infection, hematogenous or otherwise, there would have been a loss in its substance.

Histologic sections yielded the following: the tumor substance consisted of scar tissue through which there were scattered areas of round cell infiltration. Giant cells or caseation were nowhere in evidence. The picture is that if a subacute or chronic inflammation healing gradually with scar formation. The nature of the inflamed structure is merely suggested by the ring-like scar bodies in the body of the tumor, but is rendered manifest by sections of the tis-

sue at the periphery. These sections show that the scar bodies find their origin in an inflammatory process involving the glands described above, and show all stages of the process—the initial peritubular infiltration, the lysis of the gland cells, the absorption of cell substance, and, finally, the replacement of tubules by scar tissue, which on cross section have the appearance of rings. The fibres of the M. Vocalis immediately adjacent to the glands were also apparently secondarily involved. No bacteriologic studies were made of this case.

The question of differential diagnosis is in this case an interesting and difficult one. First, the possibility of an underlying tuberculous or luetic lesion as a basis for the condition must be considered; secondly, the question as to whether the chondritis was pri-

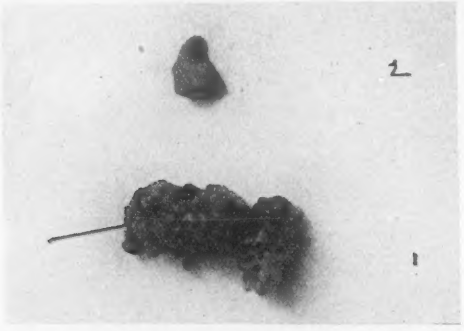

Fig. 4. (1) Gland enucleated at operation from case here described (2) Arytenoid cartilage, intact in form, found in a condition of dry necrosis, behind the tumor. The bulk of the tumor was so large as to cut off circulation of the cartilage.

mary or secondary. Acute inflammatory onset is not absolutely typical of either tuberculosis or tertiary lues. A tuberculous lesion or a gumma would have caseated and ulcerated through the mucous membrane before attaining the dimensions of the tumor. A gumma in the early stages would have rapidly responded to anti-luetic treatment. The histopathologic pictures rule out both possibilities; for, though there were nests of active inflammatory tissue interspersed in the scar tissue, there were no signs of tubercle or gumma formation. The matter of relative time of origin of the perichondritis seems to be settled by the fact that the perichondritis was in a stage of development earlier than that of the glandular lesion, as is evidenced by the absence of any signs of lysis of the cartilage sub-

stance, and the absence of pus about the necrotic cartilage, both pointing to a cutting off of blood supply by pressure of the tumor as the etiology of the necrosis of the arytenoid, and the glandular lesion as primary.

The pathology of this case differentiates it from the general category of chronic submucus laryngitides, and indicates that it represents an undescribed clinical entity—a stenosis of the larynx caused by a subacute or chronic inflammation of a group of laryngeal glands.

SUMMARY.

1. The case here presented is a laryngeal stenosis caused by a primary infection of a laryngeal structure consisting principally of a group of secretory glands, secondarily conditioning a necrosis of the arytenoid cartilage. The onset was acute, febrile, associated with a stomatitis, and the infection initially acute, subsequently ran a chronic course. The stenosis in due time became so high-grade that tracheotomy was necessary to relieve dyspnea.

2. Therapy. In view of the failure of pallative treatment, including iodides and mixed treatment, enucleation of the gland was necessitated. The gland was readily enucleated submucously.

3. The symptomatology and pathology of the case indicates that it represents a well-defined and hitherto underscribed clinical complex.

REFERENCES.

1. Bemerkungen uber den Anatomischen und Pathologischen Bau des Kehlkopfes, by E. M. Josephson—Zeitschrift fur Hals, Nasen, Ohrheilkunde, Band 2, Heft 3, page 290.

2. Traite d'histologie—A. Prenant, P. Bonin, et L. Maillard. Vol. 2. page 947.

342 Madison Ave.

X-RAY THERAPY IN CHRONIC TONSILLITIS.*

Dr. J. W. Babcock, New York.

Considerable interest in and discussion of the possibilities of X-ray therapy in chronic tonsillitis has been stimulated by a growing number of cases so treated and sometimes divergent views of the results obtained.

I beg to present some observations of myself and others and to report one of Dr. Coakley's cases by his permission, which seems to show clearly the failure of radiotherapy to produce the results attributed to it by some enthusiasts.

The shrinkage in size and sometimes the apparent disappearance, after exposure to X-ray, of lymph nodules which were enlarged as a result of some inflammation, has been repeatedly observed. Dr. F. C. Wood has expressed himself as believing that benefit might be expected where symptoms were due to simple enlargement of tonsils and adenoids. This condition, however, is one that has been doubted to exist by some authorities. Dr. W. D. Witherbee has written several articles implying favorable results of X-ray therapy on diseased tonsils and adenoids. To quote one article by him in the New York State Journal of Medicine for January, 1921, and a paper read before the American Electrotherapeutic Society in Sept., 1922, the following results may be expected from radiotherapy of diseased tonsils and adenoids: "The absorption of the lymph tissue in the tonsil, adenoid and lymph tissue of the naso-pharynx not only drains the infected crypts, but also leaves only fibrous and connective tissue, which nature utilizes in her defense in all local infections." He also gives a table showing the growth of from fifty to one hundred colonies of streptococci from the secretion expressed from the tonsils before one X-ray treatment and the growth of none from a culture made two weeks later. Reproduction of a section from a tonsil four months after one X-ray treatment is included in the article and I have taken the liberty of reproducing this for comparison with sections taken from the case to be reported in detail. Final results, according to his last statement to my knowledge, can be determined only after eight treatments at two week intervals and two months after last treatment.

In a recent article published in the Journal of the American Medical Association for February 3, 1923, I reported nine cases occur-

*Presented at New York Academy of Medicine, Section Larynology and Rhinology, April 25, 1923.

ring in the practice of Dr. Coakley and myself, who were treated by X-ray for diseased lymphoid tissue in the pharynx and nasopharynx without any improvement being noted in the finding of pathogenic bacteria in the crypts or the symptoms arising therefrom, and sections of the tonsils excised showed no change, such as is described by Dr. Witherbee. An apparent diminution in size has been noted but I wish to call attention to the fact that accurate measurements of the tonsil in situ are difficult if not impossible, and that they vary, often considerably, with the extent of inflammation then present, either with or without radiotherapy.

Waters, MacCready and Hitchcock of Johns Hopkins Hospital note a decrease in the size of chronically infected tonsils but state that there is no evidence that reduction in the size of tonsils by irradiation is accompanied by eradication of the carrier state in individuals harboring the hemolytic streptococcus in these tonsils. Dr. Sluder of St. Louis, Dr. Shambaugh of Chicago, Dr. Wood of Philadelphia and Dr. Higginbotham of Wichita have all been kind enough to write stating that their experience in radiotherapy for chronic tonsillitis had been similar to that of Dr. Coakley and myself. Several of the gentlemen of this section have also so expressed themselves. It was with the hopes that a discussion might bring forth the value or lack of value of X-ray therapy in chronic tonsillitis that this paper is presented.

Of the conceded good points of radiotherapy, its painlessness and absence of the rare but ever possible unpleasant or dangerous complications of an anesthetic, general or local, and an operation there is no question. But, if it is not effective why not seek some even more pleasant method of satisfying the desire of doing something? Then the dangers of operation on the tonsils having been thoroughly dwelt upon by the enthusiasts of X-ray therapy, why ignore the possible dangers attending X-ray therapy? There is the very real one of the wait of six months for results in certain cases of joint, heart and kidney disease, the possibility, in the hands of some less expert radiotherapists of burns, baldness, disturbance of function of salivary glands, or even sterility, and even though the parotids may be well screened, the mucous glands in the buccal and pharyngeal mucosa are not protected and in the case here reported the mouth was noticeably dry after X-ray treatment.

Case Report:

Master John M. W., age 14 years. First seen Feb. 27, 1922. Cold and talks thickly, acute inflammation adenoids and very large tonsils. Frequent colds and sore throats. He received eight treat-

ments of his tonsils and adenoids by X-ray on March 21, April 4, 15, and 29, May 13 and 27, June 6 and Nov. 14, 1922. Dec. 22, 1922, he showed acute rhinitis, tonsils still large, some secretion crypts right, moderate mass adenoids. Dec. 28, tonsils seem reduced in size about 20 per cent. Dec. 30, the culture from the left tonsil shows a growth of streptococcus viridans and staphylococcus aureus. The culture from the right tonsil shows a growth of streptococcus viridans and pneumococci. Jan. 15, 1923, tonsillectomy and adenoidotomy under gas, ether, chloroform by Dr. Coakley. The tonsils were large, buried, rather fibrous, secretion was present in crypts. They were rather adherent and difficult to separate from surrounding tissues. There was considerable lymphoid tissue in the midline of the nasopharynx and extending down behind the posterior pillars. Rosenmuller's fossae were fairly free except for some adhesions. The palate was exceptionally dry only a little sticky mucous being secreted during operation.

Microscopic Examination: By Dr. W. C. Johnson, who also kindly prepared the accompanying slides.

Both tonsils are large, the right being somewhat bigger than the left. They are covered by normal stratified squamous epithelium. The crypts are large and wide, and are lined by epithelium which appears thicker than usual. Some of the larger crypts contain masses of fungi and bacteria, others are empty. Some of the smaller branches of the crypts are filled with desquamated epithelial cells, polymorphonuclear leuocytes, lymphocytes and small masses of bacteria.

The lymphoid tissue of the tonsils is abundant, and the germinal centers are large and numerous. The germinal centers are less cellular than usual and show an increase in reticulum. Mitotic figures are present in moderate numbers.

The connective tissue framework shows no alteration. The fibrous capsule and septa are not thickened. There is no increase in connective tissue throughout the tonsils.

The adenoid tissue is abundant. It shows a more diffuse lymphoid hyperplasia than the tonsils. The crypts are small and show nothing abnormal.

Bacteriological Examination: The tonsils were held for a short time in boiling water, and then placed in a sterile dish and opened with sterile instruments. Cultures were made with a platinum loop from the deeper parts of the crypts and middle of the tonsillar tissue. Cultures on blood agar plants showed numerous colonies of hemolytic streptococci from both tonsils. The right tonsil also

showed colonies of staphylococci and the left an unidentified Gram positive bacillus. Cultures in bouillon from both tonsils showed chiefly streptococci. No culture was made from the adenoid tissue.

I beg to offer the following conclusions, that in the cases observed, X-ray therapy in chronic disease of the lymphoid tissue of the pharynx and naso-pharynx, does not cause a disappearance or fibrosis of this tissue, it does not render the crypts of the tonsil free from pathogenic bacteria, it does not relieve the patient of the pharynx and naso-pharynx[1], does not cause a disappearance or fibrosis of this tissue[2], it does not render the crypts of the tonsil free from pathogenic bacteria[3], it does not relieve the patient of acute local inflammation or the more remote symptoms due to absorption of toxins or bacteria from the tissue in question. Therefore since it does not accomplish the effects desired, it cannot take the place of adequate surgical treatment of this diseased organ, although we must concede that its ease, painlessness and freedom from the dangers of surgery must have proved very alluring to those who undertook its investigation.

The coming celebration of the fiftieth anniversary of the founding of the New York Laryngological Society, which as announced by the New York Academy of Medicine will take place November 15, 1923, commemorates an event of unusual interest. As far as can be learned this organization now the Section in Laryngology of the Academy is the oldest society in existence of the department which it represents.

In connection with the celebration there will be an exhibition representing the important contributions made to the progress of Laryngology in the city of New York.

DEDUCTIONS FROM EXAMINATION OF ONE THOUSAND ADULT THROATS.

DR. SAMUEL R. SKILLERN, JR., Philadelphia, Pa.

Having kept case records of the last one thousand adult throats especially for this paper, I was somewhat surprised in going over them to find that eighty-two per cent showed infected tonsils in one of four types. Only one half of one per cent of these cases complained of any throat condition, and twelve per cent of them gave a history of very occasional sore throat, while twenty-two per cent of them remembered having had tonsillitis, or some other form of sore throat during childhood.

The majority seemed greatly surprised when told that in all probability their tonsils were the indirect cause of their present complaints, and their stock reply seemed to be "But Doctor, I never have any trouble with my tonsils or throat". Nevertheless, if you can procure some of the debris found in the crypts of their tonsils and demonstrate it to them, they are as a rule easily convinced of their throat condition.

Four different types of tonsils seemed to prevail, and I have classified them according to the frequency of their occurrence.

1. (And I may say that this is by far the great majority.) Buried, atrophic, cryptic: the crypts of all depths.

2. Buried, normal size or hypertrophied cryptic.

3. Those that are not buried, of any size and cryptic.

4. A pin point or larger pustular infection beneath a grayish thin mucous membrane.

The majority of type four were not buried but were thin, flat tonsils of normal size or hypertrophied without crypts.

Their history as a rule was misleading, as they gave all sorts and descriptions of aches and pains from their heads to their feet, the most prevalent of which are: Nausea, chiefly upon arising in the mornings, with a thick, sticky mucus in their throats and nasopharynx which they have difficulty in getting rid of; a bad taste in their mouths; cough; no appetite; general debility, with a feeling of always being tired; boils or carbuncles; discharging ears or tinnitus; deafness, and last but by no means least, so-called rheumatic pains in any part of their bodies or extremities.

Read before the Philadelphia Laryngological Society, January 9, 1923.

Upon rhino-laryngological examination I have found in the majority of cases the following: the anterior nares are as a rule normal, occasionally the inferior turbinates will be either hypertrophied or intumescent, this often being confined to the side in which the chief infection is found in the tonsil; the posterior nares shows a low grade inflammation of the entire mucous membrane extending down as far as can be seen by nasal inspection; often the nasopharynx contains a glary, sticky, colorless secretion. Upon examination of the throat your attention is at once directed to the anterior pillars which are sometimes swollen and injected, at other times merely show a low grade of inflammation. Occasionally the anterior pillars will appear to be normal. This is exceptional.

The Plica-Tonsillaris is generally edematous and often hypertrophied, the amount of this depending to a great extent upon the duration of the infection in the tonsils. I have seen any number of cases in which the plica entirely obscured the tonsils, due to enlargement, and often adhesions to the tonsillar or surrounding tissue. In several cases in which the old guillotine operation had been done a number of years before, the plica not only covered but had become so intimately connected with surrounding tissue that I had to split it with a bistoury before the tonsils could be examined. Numerous cases showed a very small portion of what appeared to be normal tonsil which, when the plica was split, showed infection in other parts of the tonsil.

In this type of case care must be taken to get the pillar retractor well around the plica-tonsillaris, engaging the anterior pillar and pulling it forward, at the same time pushing outward and backward against the tonsil which, with the gagging of the patient, will throw the tonsil inward out of the bed, thus fully exposing it and at the same time expelling any secretion or collection of debris that may be in the crypts or between the anterior pillar and the tonsil.

If no pus or debris is found by this method I then use what I consider a rather clever method first shown me by Dr. George M. Coates, of making sure that the crypts are not infected at their base, which mere pressure sometimes fails to bring out.

A metal or hard rubber Eustachian tube catheter is attached to the compressed air cock. Holding the tongue well down, the mouth of the catheter is held about ¼ inch from the opening leading down into the crypt. Using about 25 pounds of air pressure, the air is blown into the crypts in short puffs. This will dilate the crypt and its contents will be forced out, often with such force that the contents will fly across the mouth or even out of the mouth striking

the operator in the face. This method, as far as I have been able to ascertain completely empties the crypt, and I often use it in cases that refuse operation or in which operation is contra-indiciated. This procedure in treatment gives, of course, only temporary relief, as the crypts will soon fill, but it does remove the source of infection for the time being and, in connection with antiseptics applied to the tonsil, will keep down the infection.

I wish to emphasize the fact that the catheter must be held a short distance from the mouth of the crypt, that others may not make the mistake that I did. If the catheter is placed in the mouth of the crypt and air suddenly forced through it, you are very apt to force the air not only into the crypt but through it into the peri-tonsillar connective tissue, and if the crypt be infected the infection may be carried along thus causing a peri-tonsillar abscess, or Quinsey. If the crypt be clean no more harm is done than to cause a circumscribed area of emphysema which is somewhat alarming to the patient and more or less of a discomfort until it disappears.

As I stated, the tonsils were generally of the atrophic, cryptic variety, but I was often surprised upon inversion to find them of normal size or hypertrophied due to their being deeply buried, giving them the appearance of small normal tonsils when first examined. I bring out this point to show the great importance of retracting the pillars in all tonsil examinations.

The crypts may be either shallow clefts or deep tortuous canals, and what often appeared to be clean crypts would upon pressure give up a grayish white or pure white, foul smelling, cheesy mass consisting of putrifying food and other debris from the mouth, and almost every kind of bacteria. Often free pus alone would pour out.

The oro-pharynx showed the same type of low grade inflammation as the naso-pharynx, but of a more severe grade and often covered with the same glary, colorless secretion. As shown by the grades of inflammation extending from the tonsils to the nares, it is easily seen that the inflammation started in the tonsils and gradually fades out the farther away it gets.

Either post-nasal (by means of the naso-pharyngoscope) or post-oral (by means of the laryngeal mirror) examination reveals the same type of inflammation about the swollen orifices of the Eustachian tubes.

The majority of men suffering with chronic purulent otitis media, and often times those with a simple catarrhal otitis media, have one of the types of cryptic infected tonsils I have described. But why

shouldn't they? When we stop to consider that the same type of mucous membrane covering the tonsils extends backward and upward into the oro and naso-pharynx, the nose and sinuses, the Eustachian tubes, entire middle ear, and even into the attic and mastoid cells, we can readily see why the infections from the tonsils can travel to any or all of the above cavities where they find ideal place for growth.

We all know that bacteria will spread on the type of membrane its growth started on in preference to changing to a membrane of a different type. Hence we get a sinusitis, or salphingitis, or otitis from infected tonsils when a much more direct and apparently fertile field would be the lower respiratory tract.

The Eustachian tubes are in close proximity. When we stop to consider that every time we swallow or suddenly force air through our noses, as in blowing the nose, we open the Eustachian tubes, forcing air and whatever secretion, whether it be sterile or infected, that may be covering or in proximity to the orifices back into the tubes, we can see what a simple matter it is for infection to gain entrance, not only to the ear but even the mastoid cells.

But why all this reiteration of facts that we all know? Merely this! That if we can take the data which I have compiled as an average for the men of this locality between the ages of twenty-five and fifty, then eighty per cent of them are carrying around not sources of infection but a true infection, which is lowering their body resistance to other infections, as well as causing them all kinds of vague symptoms, with hidden pathological lesions throughout their entire systems.

There is at the present time a great difference of opinion as to the advisability of removal of infected tonsils in pulmonary tubercular cases. My experience in the Philadelphia General Hospital, while on tubercular service, and from post-mortem examinations while on pathological service, showed me that the great majority of all pulmonary tubercular cases died of some intercurrent infection, generally pneumonia, and not tuberculosis per se. If, as I think, the the general source of opinion shows this to be true, why ask the poor tubercular case to carry around a cess-pool in his throat which is not only lowering his resistance to the tubercle-bacillus by pouring poisons into his system, but also endangering him to a mixed infection at any time? Doesn't the tubercular case have a hard enough fight without asking him to carry this extra burden? I have heard the theory expounded that a person is more susceptible to colds after removal of the tonsils, and this by physicians. In my

opinion this is all rot. Have the tonsils ever been proven to have any protective action to the throat? It is true we may get an acute exacerbation of a latent infection after their removal as seen so often in so called rheumatism. But after this acute exacerbation is controlled there is always a marked improvement.

With due care to have the tubercular patient fit for operation and with careful instrumentation and after treatment, I personally feel that every tubercular case that is not too far advanced can reap nothing but benefit from the removal of his or her infected tonsils.

Having the patient fit for operation and with a skillful operator, what harm can possibly be done by removal of the tonsils under *local* anesthesia? I emphasize local anesthesia, as I do not believe any pulmonary condition should be irritated by the inhalation of any form of general anesthetic. With proper technic, using as little as possible of apothisin and adrenalin, but being sure you have proper anesthesia to avoid shock, using the LaForce instrument when possible or a careful dissection of the tonsils with ligation of the tonsillar artery, if necessary, and removal of all secretion by continual suction and instruction to the patient to avoid inhalation of any fluid, the after-treatment of one per cent (1%) cocain sprayed on the throat before each liquid feeding until the patient is able to swallow with some degree of comfort, and absolute rest in bed for two or more days, can in no way be of anything but a future benefit to the case.

TO SUMMARIZE.

1. Apparently 80 per cent of men in this locality have infected tonsils.

2. Infected tonsils are the cause of the majority of cases of so-called rheumatism, naso-pharyngitis and otitis media, and are often the indirect cause of sinusitis.

3. Incipient tuberculosis and so-called early tubercular cases, if in otherwise good physical condition should have their tonsils removed provided they are infected or of the cryptic type.

2008 Walnut Street.

TREATMENT OF ACUTE AND CHRONIC DACRYOCYSTITIS WITH SILVER CANNULAS.

DR. JOHN F. CALLAHAN, Brockton, Mass.

It is not within the province of this paper to impose a description of the structural anatomy of the lachrymal apparatus nor the pathology and causation of the condition treated. I am attempting to bring to your attention a simple surgical principle in the draining of an abscess and the reduction of a stricture in a bony canal without any anatomical disfigurement and with the least amount of inconvenience to the patient. A volume would be required to give in detail an account of the various operative procedures which have been proposed from time to time for the cure of stricture of the lachrymal duct or a relief of the connsequences to which it gives rise. As this paper has to deal with a metal cannula, gold or silver, I will attempt to limit my discussion of the subject of lachrymal drainage of the present and past use of metal probes, cannulas, and styles.

The cannula which I offer in this paper for the treatment of acute and chronic dacryocystitis has not, so far as I can find in medical literature, been used according to the technique that I have devised. The cannula as seen in cut consists of tubes of gold or silver, the walls of which are about .005 of an inch in thickness. The essential thing is that they shall be pliable enough so that they may be passed into the slightly curved nasal canal and withdrawn by way of the nasal end beneath the inferior turbinate. They must also be of a sufficient rigidity so that they will not slip through into the nose without being manipulated. To accomplish this, my cannula is specially annealed.

In my early attempts to find a suitable cannula, I used a spring cannula of German silver but on two occasions the spring cannulas worked into the nose and the patients swallowed them. Another point against the spring cannula is that like all metallic springs of the metal and weight that we could use for this purpose, they are brittle and are liable to break while being removed. Also, the fact that they will tend to work into the nose, has caused me to change in favor of the simple silver tube.

Wathen in England in 1781 first used a cannula of gold or silver in the treatment of stricture of the lachrymal duct. The cannula used by Wathen was introduced through a divided canaliculus and

its flange-shaped upper end prevented its falling through the duct into the nose or being removed by the nasal route.

Various attempts have been made to cure stricture of the nasal duct by passing thread into canal and out through the nose.

Several years ago when probing and styles were the order of the day, I made many unsuccessful attempts to treat stricture of the nasal duct by probing and the constant wearing of styles. I used Bonman probes and styles up to No. 12 and I cannot say that I ever registered a cure. After a perusal of Theobald's[1] Article 1, I started using larger sized styles, 13 to 16. I occasionally see cases that I used the large sized styles on ten to thirteen years ago and they still have patent nasal ducts. I never could report a permanent result where I used a style that was less than 3¼ mm. in diameter. The only permanent results that I recorded were in cases that I used styles of over 3 mm. in diameter and left the style in place for at least three months. In view of this experience, the smallest cannula that I use is 3¼ mm. in diameter and I always place the largest sized cannula in nasal duct that can be introduced without force. Theobald says that in a review of his cases including those met in children, the No. 16 or the 4-millimeter probe had been used in about 66 per cent of his cases.

TREATMENT OF ACUTE DACRYOCYSTITIS.

Verhoeff[2] is credited with the return to practise of Agnew's[3] operation which had been overlooked or forgotten for some forty-five years. Agnew's operation consists of an incision made through the conjunctiva between the caruncle and the inner commissure of the eye lids. It involves cutting through the least possible depth of tissue in this condition where the tissues of the face about the lachrymal sac are usually swollen. The knife is plunged into the sac at a point between the caruncle and the inner commissure of the lids, the incision being carried through the sac until we feel the knife strike the bone. The incision is then enlarged by cutting upwards and outwards. On alternate days following the operation, the duct is probed through the incision and in some cases the probing may have to be continued for a period from one to three weeks.

Agnew's operation is now in use in a great many clinics but some still favor the skin incision down to the sac which opening requires a wick and, soon after its removal, the wound often has to be opened and wicked again.

Anyone with any experience in the treatment of this condition well knows the horror the patient has of the persistent probing in the Agnew operation. This treatment at its best is merely for tem-

porary relief of the acute abscess and I have never heard anyone claim that the stricture of the nasal duct—which is always the cause in these cases that occur during the course of a chronic dacryocystitis—was reduced and nasal duct made permeable again. The sometimes futile attempt to drain by the skin route which often ends in a chronic fistula due, of course, to the non-surgical principle in-

volved in attempting to drain an abscess cavity from its upper limits as the wick never reaches the lower limits of the abscess. In placing the cannula in the duct, I first used the route proposed by Agnew if a fistula was not already present. In the last two cases in which I have used the cannula in acute dacryocystitis, I have entered the sac through the skin as a fistula was present and it appears to me as the easiest means of approach for, with the greatly swollen lids,

considerable force has to be used in some cases with Agnew's incision whereas, by the skin route, we just plunge the knife right into the abscess and there is apparently less trouble in passing the probe and cannula into the lachrymal sac and down into the nose.

When the silver cannula is introduced at the opening of the abscess our work is completed. We have followed one of the first principles of surgery; we are draining an abscess in a bony canal from the bottom. It is really remarkable the relief that is given the patient. There is immediate and enduring relief from the pain. Probing and other instrumentation are no longer required. It is not necessary to use another instrument in the treatment until we remove the cannula by way of the nose at the end of three months. The cannula method in acute dacryocystitis will not only clean the case up quickly and with less instrumentation and with less discomfort to patient than any procedure that I have attempted, but it also affords a possibility of a permanent cure for the stricture of the nasal duct. The method of approach when the cannula is used in chronic purulent dacryocystitis is by Agnew's route as we find that there is less trouble in this condition in getting into the conjunctival sac where the incision is made than in the acute form.

TECHNIQUE REQUIRED FOR SELECTION OF LENGTHS AND DIAMETER OF TUBES AND THEIR INTRODUCTION INTO THE LACHRYMAL CANAL.

The length of the tube may be determined from external measurements; a rule may be placed externally on face and measurements taken from orbital margin anterior to lachrymal sac to a point on level with floor of nose beneath lower turbinate. As the stricture is usually confined to the nasal duct, our measurements need not be exact but the tube should be made long enough to extend well above nasal duct but always below a level of the orbital rim and when in position should never extend high enough to be felt by palpation.

After incision into lachrymal sac has been completed, the largest probe that is possible is passed through canal into nose. The next size smaller tube than the probe that was introduced is then selected as one that will easily allow its introduction into and through canal. At the present I am using only two cannulas. The largest cannula has an outside diameter of four millimeters and the smaller cannula three and one quarter millimeters. I have constructed probes patterned after Ziegler's lachrymal dilators which fit snugly in the cannulas and act as obturators, the nasal end of the cannula being tappered to conform with the obturator. Cannulas are one and three quarter inches long and may be cut to the required measurements to suit the case as the lengths required in different lachrymal canals

vary from three quarters to one and three quarter inches. Cannulae are easily cut with scissors. Each cannula has three holes each two millimeters in diameter near the nasal end as it is possible that the nasal end of cannula may settle into mucus membrane at floor of nose thus obstructing the lower end. The patient never has any discomfort when wearing tube and doesn't even feel it there.

REMOVAL OF CANNULA AT END OF THREE MONTHS.

Four or five drops of 4 per cent solution cocaine are instilled into conjunctival sac and a 10 per cent solution cocaine with adrenalin is applied in region of lower turbinate. A mosquito hemostat is passed beneath lower turbinate and closed over lower end of cannula and hemostat is gently rotated and cannula which collapses under slight pressure is wound around hemostat and removed from nasal cavity. There is little discomfort caused in removal and I have not, as yet, been required to use a general anesthetic. The silver cannulas, when removed, were very clean with no deposits in interior or outer wall. The only attempt at cleaning cannulas was with argyrol, silvol and zinc sulphate solutions dropped into conjunctival sac. The relief that is given in the acute cases is remarkable. My first case of the chronic form was tubed December 24, 1921 and the tube left in the canal for three months. Patient has had no epiphora and argyrol instilled into conjunctival sac readily passes into nose. Patient suffered from chronic purulent dacryocystitis for years. To April, 1923, I have placed cannulas in ten cases of which four were acute and six chronic. All cases are free from pus and only one still suffers from epiphora, this latter case being due to obstruction in canaliculi. The antiseptic action of the silver cannulas is possibly an aid in abating the purulent process.

I presume I may also state I failed in one case. The hemorrhage in this case was quite severe and I was unable to see anything in the nasal cavity. Three days later I examined patient's nose and found cannula extending from sac into nose in front of anterior end of middle turbinate. Patient recovered without further operative procedure from her acute dacryocystitis. Three months later patient suffered another attack of acute dacryocystitis and submitted to operation. April 13, 1923 I inserted a cannula in each lachrymal sac via Agnew incision as she had an affection of both lachrymal sacs. This patient had been an asthmatic for eighteen years, having an attack every night. June 13, two months after cannulas were inserted, patient reports that she has been entirely free from asthma since cannulas were inserted and is of the opinion that the silver tubes are the cause of her relief but I am of the opinion that the drainage of the purulent process is the best guess.

The limited number of cases that one sees in a city the size of Brockton is not sufficient for such an experiment as it would require years before one would be able to collect data on the results. I do think that there are possibilities in this method that none of the other procedures have shown and hope that some one with a large amount of clinical material will give the cannula a trial.

CONCLUSION.

Operations, external and internal, as demonstrated by Berlin, Toti, Mosher, Chamberlain, West, and others, are still on trial as years alone will judge their success or failure. Extirpation of the sac has evidently fallen into disrepute as it always failed in relieving the patient of the epiphora. That results have been obtained in probing, no one will doubt. All that was necessary was the faithful attendance of the patient. Styles are objected to as it is not considered ideal to slit the canaliculus together with the fact that it is impossible to estimate the exact length as the measurements have to include the soft tissues as well as the bony canal to the floor of the nose. To place a silver or gold cannula in the lachrymal sac consists of one simple operation, that is, passing the proper sized probe into the canal.

While I leave the tubes in three months, we could just as well leave them in indefinitely. There is no scar or deformity of parts and the pressure of the light silver tube should cause an ideal condition and place the sac in as near a normal condition as it is possible after being exposed to a purulent process over an extended period.

Plate shows silver cannula that had been in patient three months. This shows that cannula did not slip from its original position and is resting on that portion of the floor that curves up to form the lateral wall of antrum.

REFERENCES.

1. THEOBALD: Diseases of the Lachrymal Apparatus. Norris and Oliver. Volume 3, page 133.

2. VERHOEFF: *Journal American Medical Association*, CX, page 727, 1913.

3. AGNEW: *American Practitioner*, January, 1817.